

DUPLICATE

TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

TENTH SESSION

HELD IN CINCINNATI, JUNE 1, 2, AND 3, 1898.

WITH AN INDEX OF VOLS. I TO X.

EDITED BY

FLOYD M. CRANDALL, M.D.

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B. K. RACHFORD,	C. P. PUTNAM.

MEETING PLACES.

1888.	WASHINGTON (Organization), September 18.
1889.	WASHINGTON and BALTIMORE, September 20 and 21.
1890.	NEW YORK, June 3 and 4.
1891.	WASHINGTON, September 22 and 25.
1892.	BOSTON, May 2, 3, and 4.
1893.	WEST POINT, May 24, 25, and 26.
1894.	WASHINGTON, May 29 and June 1.
1895.	VIRGINIA HOT SPRINGS, May 27, 28, and 29.
1896.	MONTREAL, May 25, 26, and 27.
1897.	WASHINGTON, May 4, 5, and 6.
1898.	CINCINNATI, June 1, 2, and 3.
1899.	DEER PARK.

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A. JACOBI.

Alternate, - - - - A. CAILLÉ.

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DORNING, JOHN, M.D.....	252 West Twenty-fifth Street, New York
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FRUITNIGHT, J. HENRY, M.D.....	161 West Fifty-seventh Street, New York
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HAMILL, SAMUEL McC., M.D.....	1822 Spruce Street, Philadelphia
HOLT, L. EMMETT, M.D.....	14 West Fifty-fifth Street, New York
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MCCOLLOM, F. H., M.D.....	745	Massachusetts Avenue, Boston
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MORSE, J. LOVETT, M.D.....	317	Marlborough Street, Boston
MUSSER, JOHN H., M.D.....	1927	Chestnut Street, Philadelphia
NORTHROP, WILLIAM P., M.D.....	57	East Seventy-ninth Street, New York
OSLER, WILLIAM, M.D.....	1	West Franklin Street, Baltimore
PACKARD, FREDERICK A., M.D.....	110	South Eighteenth Street, Philadelphia
PUTNAM, CHARLES P., M.D.....	63	Marlborough Street, Boston
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STARR, LOUIS, M.D.....	1818	S. Rittenhouse Square, Philadelphia
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VAUGHAN, V. C., M.D.....	15	South State Street, Ann Arbor
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WILLIAMS, HAROLD, M.D.....	528	Beacon Street, Boston
WILSON, J. C., M.D.....	1437	Walnut Street, Philadelphia
WINTERS, J. E., M.D.....	25	West Thirty-seventh Street, New York
YALE, LEROY M., M.D.....	432	Madison Avenue, New York

██████████

Deceased.

██████████

JOHN A. JEFFRIES, M.D.,
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.,
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.,
Born, April 30, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.,
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.,
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.,
Born, October 12, 1841,
Died, January 7, 1898.

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MINUTES OF THE TENTH ANNUAL MEETING OF THE
AMERICAN PEDIATRIC SOCIETY.

Held at Cincinnati, June 1, 2 and 3, 1898.

The following members were present:—G. N. Acker, M.D., Washington; S. S. Adams, M.D., Washington; A. Baines, M.D., Toronto; W. D. Booker, M.D., Baltimore; E. Buckingham, M.D., Boston; A. Caillé, M.D., New York; W. S. Christopher, M.D., Chicago; F. S. Churchill, M.D., Chicago; F. Forchheimer, M.D., Cincinnati; R. G. Freeman, M.D., New York; J. P. C. Griffith, M.D., Philadelphia; L. E. Holt, M.D., New York; F. Huber, M.D., New York; A. Jacobi, M.D., New York; C. G. Jennings, M.D., Detroit; C. G. Kerley, M.D., New York; J. L. Morse, M.D. Boston; W. P. Northrup, M.D., New York; B. K. Rachford, M.D., Cincinnati; T. M. Rotch, M.D., Boston; I. M. Snow, M.D., Buffalo; J. P. West, M.D., Bellaire; J. E. Winters, M.D., New York; J. H. Fruitnight, New York.

FIRST SESSION.—JUNE 1.

The meeting was called to order by the President, Dr. L. Emmett Holt, of New York.

Dr. Francis Huber, New York, reported a case of "Congenital Sacro-Coccygeal Tumor."

Discussion by Dr. Jacobi.

Dr. F. Forchheimer, Cincinnati, read a paper entitled the "Exanthem of German Measles."

Discussion by Drs. Griffiths, Northrup, Jacobi, Buckingham, Adams, Rotch, Caillé and Fruitnight.

Dr. F. S. Churchill, Chicago, read a paper entitled "The Urine of Healthy Infants and Children."

Discussion by Drs. Christopher, Tyson, Fruitnight, Rotch and Griffith.

Dr. Charles G. Kerley, New York, read a paper on "Acute Nephritis of Malarial Origin in Early Childhood."

Discussion by Drs. Northrup, Caillé, Fruitnight, Forchheimer, Adams and Jacobi.

Dr. B. K. Rachford, Cincinnati, read a paper on "Albuminuria Accompanying Lithæmic Attacks."

Discussion by Dr. Caillé.

Dr. J. L. Morse, Boston, read a paper on the "Classification of the Anæmias of Infancy."

Discussion by Drs. Morse and Freeman.

SECOND SESSION.—JUNE 2.

Upon motion the following letter was sent to Dr. John A. Larrabee, of Louisville:

"The American Pediatric Society learn with regret of the serious illness of Dr. John A. Larrabee, and desire to express its sympathy for him and the hope that the life of so prominent a worker in diseases of children may be long spared."

The report of the Committee of the Society on the Collective Investigation of Scurvy in America was read by the Chairman, Dr. Griffith.

It was moved that the thanks of the Society be expressed to the Committee for their work. This was seconded and unanimously carried.

Discussion by Drs. Caillé, Jacobi and Griffith, upon the motion to accept the report of the Committee.

Discussion upon the report by Drs. Christopher, Griffith, Northrup, Morse, Jennings, Booker, Caillé, and Forchheimer.

During the discussion Dr. Caillé submitted a Minority Report.

On motion, the report was referred back to the Committee with instructions to report on the following day.

Dr. T. M. Rotch, Boston, presented short reports on "Some Unusual Cases."

(a) Two cases of Melanosis Lenticularis Progressiva.

(b) Two cases of Intussusception.

(c) A case of Cerebro-Spinal Meningitis.

Discussion by Drs. Huber, Fruitnight, Winters, Northrup, Conner, Jacobi and Caillé.

Dr. George N. Acker, Washington, reported "Cases of Tubercular Pyelitis" and "Death from Pulmonary Hemorrhage in an Infant Aged Two Years."

Dr. F. S. Churchill, Chicago, reported a case of "Sarcoma of the Kidney in an Infant Aged Nine Months."

Discussion by Drs. Morse, Jennings, Jacobi and Fruitnight.

THIRD SESSION.—JUNE 2.

The annual address of the President, entitled "Scope and Limitations of Hospitals for Infants," was read by Dr. L. Emmett Holt, New York.

Dr. W. S. Christopher, Chicago, read a paper entitled "The Fatigue Period in Child-life."

Discussion by Drs. Jacobi, Forchheimer, Rotch.

Dr. C. G. Kerley, New York, reported "Seven cases of Laryngeal Diphtheria Treated with Antitoxin, with One Death."

Discussion by Drs. Caillé and Fruitnight.

Dr. A. Jacobi, New York, reported "Three Cases of Amurotic Idiocy."

FOURTH SESSION.—JUNE 3.

A discussion on the question "Should all Milk used for Infant Feeding be Heated for the Purpose of Killing Germs? If so, at what Temperature, and How Long Should this Temperature be Continued," was opened by Dr. Freeman, of New York.

The revised report of the Committee on Scurvy was presented by Dr. Griffith.

Discussion on Dr. Freeman's paper and the report by Drs. Booker, Buckingham, Winters, Morse, Jacobi, Adams, Caillé, Jennings, Griffith, Holt, and Freeman.

Dr. Caillé, New York, read a paper entitled "The Significance of Fever after Operations for Pyo-Thorax."

Discussion by Dr. Rotch.

Dr. Adams, Washington, read a paper entitled "Irrigation by Submersion in the Treatment of Empyema."

Discussion by Drs. Winters, Caillé and Jacobi.

Dr. Snow, Buffalo, reported "Two Cases of Insolation in Infants."

The following papers were read by title: "A Dermoid Cyst of the Large Fontanelle," by A. Jacobi.

"A Study of an Epidemic," by W. L. Carr.

"Clinical Observations in Tubercular Meningitis," by H. D. Chapin.

"A Case of Congenital Defect of the Biliary Duct," by C. P. Putnam.

"Peculiar Types of Broncho-Pneumonia," by F. M. Crandall.

BUSINESS MEETING.

On nomination of the Council the following Officers were elected:

<i>President,</i>	-	-	-	-	-	DR. W. P. NORTHRUP.
<i>First Vice-President,</i>	-	-	-	-	-	DR. GEO. N. ACKER.
<i>Second Vice-President,</i>	-	-	-	-	-	DR. IRVING M. SNOW.
<i>Secretary,</i>	-	-	-	-	-	DR. SAMUEL S. ADAMS.
<i>Treasurer,</i>	-	-	-	-	-	DR. E. M. BUCKINGHAM.
<i>Recorder and Editor,</i>	-	-	-	-	-	DR. FLOYD M. CRANDALL.
<i>Delegate to the Congress of American Physicians and Surgeons,</i>						DR. A. JACOBI.
<i>Alternate,</i>	-	-	-	-	-	DR. A. CAILLE.
<i>Member of Council,</i>	-	-	-	-	-	DR. F. FORCHHEIMER.
<i>New Members,</i>	DR. A. C. COTTON, Chicago; DR. D. J. MILTON MILLER, Philadelphia; DR. S. M. HAMILL, Philadelphia.					

Place of Meeting, *Deer Park Hotel, Maryland.*

Time of Meeting, *June—, 1899.*

It was voted that the Transaction of the Society be printed as last year, and under the same conditions. An appropriation to cover additional expenses for publishing the Transactions for 1897 was made, and also an appropriation to defray the expenses incurred by the Collective Investigation Committee. An assessment of Five Dollars was made for annual dues. The Treasurer submitted a report showing a balance of \$146.06. It was moved and carried that reprints should be sent to the contributors of the Scurvy Report and that they should contain both the majority and minority reports and the discussion.

A vote of thanks was passed to the resident members for their kind attentions.

FLOYD M. CRANDALL, M.D.,

Recorder.

THE AMERICAN PEDIATRIC SOCIETY'S COLLECTIVE INVESTIGATION ON INFANTILE SCURVY IN NORTH AMERICA.

The subject of infantile scurvy has so recently come into prominence, and still presents so many mooted questions, especially regarding its etiology, that it was the decision of the American Pediatric Society a year ago to undertake a collective investigation of the matter, based upon the cases occurring in America. This seemed particularly needed, as no other such study upon a large number of cases has yet been made in any country.

The committee, which is now making its report, was accordingly appointed. It has been diligently at work during nearly a year, and has used every means in its power to reach reports of cases of the disease. A list as accurate as possible was prepared of all the medical journals of North America, and a notice of the proposed investigation was sent to each, inviting correspondence on the part of all readers. Letters were sent to the secretaries of the county societies in a large number of the States of the Union requesting that notice be given at the meetings. Letters were also addressed to the professors of diseases of children in all the regular medical colleges of the United States. The "Index Medicus" was searched for the names of those who had published reports of cases, and letters were addressed to all of them, as indeed to all physicians of whom even the rumor had come of probable cases under their charge. Circulars were printed containing questions to be answered, and were sent to all the members of the American Pediatric Society, to all physicians applying for them, and finally wherever there seemed any chance of getting a response.

The questions contained in the circular requested information on the following points: Whether the case was seen in hospital

or private practice; the race, sex, and age; the hygienic surroundings, family history, and previous illnesses; full details of feeding from birth, and the influence which the food appeared to have had upon the development of the disease; the symptoms in detail, with special reference to pain and its location, apparent paralysis or inability to move, swellings, fractures, hemorrhages, the condition of the gums, the presence of fever, the condition of the urine and bowels, the presence of anæmia or mal-nutrition and of rickets or any other complicating diseases, and the character of the first symptom to develop; the treatment in detail, with duration of illness, and the time before decided improvement was discovered; the direct cause of death in fatal cases, and the *post-mortem* findings; and finally, whether the case had been published previously.

The committee has been surprised and pleased at the large number of replies received. There are other cases of which it has knowledge of which no reports could be obtained, and undoubtedly many more whose existence was not discovered. But in all the committee has collected 379 cases seen by 138 observers. Some of the cases are very incompletely reported, but in the majority of instances the answers are for the most part satisfactory. No cases needed to be excluded as instances of mistaken diagnosis, although a very few were somewhat doubtful.

The topics covered by the questions can best be taken up for the most part seriatim, stating merely what the reports state, without vouching for the correctness of opinions.

RACE.—The race to which the subjects of the disease belong is stated in 372 cases. It is not given with definiteness sufficiently often to allow of an analysis further than to say that there were 367 white, 4 black, and 1 Chinese.

SEX.—Sex shows out of 372 cases, 189 male, *i.e.*, 51 per cent.; and 183 female, *i.e.*, 49 per cent.; a difference not decided enough to indicate that sex is an etiological factor. In the remaining cases the sex is not mentioned.

AGE.—Age is a very important matter. Although strictly speaking, the age of the cases of infantile scurvy should be limited to those under two years, the committee has ventured to include a few in children slightly or decidedly older than this; since the etiology and symptoms are not different in any respect. Question IV. on the circular reads: "Age when seen

with Scurvy," while XIV. reads: "Duration of Illness Before Treatment was Commenced." By combining the answers to these two questions, the age at which the illness developed could be determined in most instances. Reliable information was given in 359 cases; in the remaining number the age was unknown or was not stated. The accompanying tabular arrangement shows the number of cases developing at different ages:

AGE WHEN SCURVY DEVELOPED.

AGE.	NO. OF CASES.	PER-CENTAGE.	AGE.	NO. OF CASES.	PER-CENTAGE.
3 weeks,	1	.27	16 months,	7	1.70
1½ months,	1	.27	17 "	6	1.67
2 "	3	.83	18 "	7	1.70
3 "	2	.55	19 "	4	1.11
4 "	9	2.50	20 "	2	.55
5 "	5	1.40	22 "	1	.27
6 "	13	3.62	23 "	1	.27
7 "	33	9.19	2 years,	2	.55
8 "	41	11.42	2 years, 3 mo.	1	.27
9 "	47	13.09	2 " 6 "	1	.27
10 "	51	14.20	2 " 7 "	1	.27
11 "	26	7.24	2 " 8 "	1	.27
12 "	25	7.00	3 " 6 "	1	.27
13 "	25	7.00	4 " 2 "	1	.27
14 "	22	6.12	6 "	1	.27
15 "	17	4.73	9 "	1	.27

It will be seen that the disease is most apt to develop between the ages of seven and fourteen months, inclusive. The youngest case was that reported by Dr. A. Matheson of Neillsville, Wis. This was a child of four weeks who had already been ill five days when first seen. The child was fed at the breast. Its hygienic surroundings were poor. The disease exhibited perfectly typical symptoms and ended fatally.

The oldest child, reported by Dr. J. H. Fruitnight, was one of nine years, and was also a typical case, rapidly recovering under dietetic treatment. The cause appeared to be improper diet.

SOCIAL POSITION.—Of 379 cases it is interesting to note that 83 per cent. occurred in private practice, and only 17 per cent. in hospital practice. Although no absolute class distinctions can be based on these figures, yet they point very positively to the greater tendency of the disease to occur among the rich or the well-to-do. This tendency is still further illustrated by the statements of the writers regarding the *hygienic surroundings* of

the cases. In 303 cases these are described as good, and often the statement is volunteered that they were of the very best. In 5 they were doubtful, and in only 40 are they described as bad. The figures of the report would therefore seem to indicate that the influence of bad hygienic conditions upon the etiology of the disease is extremely limited.

PREVIOUS HEALTH.—Out of 285 cases suitable for study, it is distinctly stated in 167 that the previous health had been good. In 118 the child had suffered from various diseased conditions which may be enumerated as follows:

Bronchitis, 5; chickenpox, 1; constipation, 1; convulsions, 3; cretinism, 1; diarrhœal conditions, 45; eczema, 1; furunculosis, 1; indigestion, 22; influenza, 6; malaria, 1; measles, 7; pneumonia, 5; rheumatism, 1; scurvy (previous attack), 1; scrofulous diathesis, 1; typhoid fever, 1; whooping cough, 2.

It is evident that the occurrence of most of these diseases can only be considered as accidental. There is a striking preponderance of instances of digestive disturbance. This probably is an indication that the faulty diet which occasioned the scurvy produced the indigestion also. It is no proof that the digestive disease itself bore any etiological relation to the constitutional affection. This is clearly the view of the correspondents, for, in answer to the question of the circular, a belief in any other cause than diet is expressed in only 24 instances.

Rickets, anæmia, and mal-nutrition are not mentioned in the foregoing list. They will be referred to later.

Attention may be called to the instance of a second attack of scurvy reported by Dr. L. E. Holt. The child was eighteen months of age at the time of the second attack, the previous one having developed four months before. The first attack followed the use of Mellin's food and sterilized milk. Recovery followed in a week upon a diet of sterilized milk and beef juice, no fruit juice being given. The second attack followed the use of Reed & Carnrick's soluble food. The patient in this attack was in a wretched condition and died in eight days.

The case of scurvy developing in a cretin, reported by Dr. A. Caillé is also interesting. The child was a typical cretin of fourteen months. Scurvy followed the use of condensed milk. Recovery was very prompt under the administration of sterilized milk, fruit juice and cereals.

FAMILY HISTORY.—This too appears to exert little or no influ-

ence. In 129 cases the family history is stated to have been good, and in 97 it is negative. In 74 the following diseases are mentioned in the family:

Alcoholism, 2; anæmia, 2; asthma, 1; carcinoma, 1; caries of spine, 1; diarrhœa, 1; eczema, 1; gout, 2; neurotic tendency, 6; paresis, 1; pneumonia, 1; rheumatism, 16; sciatica, 1; scurvy, 1; syphilis, 7; tuberculosis, 29; uricacidæmia, 1.

DIET.—The most important etiological factor, according to general opinion, is a dietetic one. Consequently, the committee has paid particular attention to this point. When correspondents did not make the matter quite clear in their answers, personal letters were addressed to them, asking for further information. A large number of such letters have been written, and replies received in most instances. Full details were asked regarding diet from birth onward, and the question of food used at the time the scurvy developed, or so shortly before that it might seem to be associated with it, was particularly emphasized. The question was also asked, whether in the opinion of each correspondent there was reason to believe that the disease depended on the nature of the food used. An affirmative answer was received in 275 cases; negative, and the disease attributed to other causes, in 24. The committee is not in a position to judge of the correctness of this view, nor can it claim that the disease *did* arise in any instance as the result of the diet employed. It would make merely the following statements of the food employed at or shortly before the symptoms of scurvy were observed, according to the reporters' replies.

Any accurate percentage analysis of the report is impossible, both because the correspondents have not always stated the exact nature of the food, and because in very many instances more than one form of food was given. Perhaps the following summary of some of the main divisions may be of value, remembering, however, that cases are repeatedly counted twice; *e.g.*, one case may be counted in the condensed milk class and again in the sterilized milk division.

FOOD USED AT OR SHORTLY BEFORE SCURVY DEVELOPED.

Number of cases in which the character of the food is specified, 356.

Food given as follows:

Breast Milk.—Alone, 10; with raw milk and amylaceæ, 1; with sterilized milk and amylaceæ, 1; total, 12.

Raw Milk.—Alone, 4; with breast milk and amylaceæ, 1; total, 5.

Milk (nothing said about heating).—Alone, 8; peptonized, 4; with amylaceæ, 4; total, 16.

Sterilized Milk.—Alone, 68; with proprietary foods, 21; with amylaceæ, 8; peptonized, 10; total, 107.

Pasteurized Milk.—Alone, 16; with proprietary foods, 2; with amylaceæ, 1; peptonized, 1; total, 20.

Peptonized Milk.—Nothing further stated, 3; sterilized, 8; pasteurized, 1; with proprietary foods, 1; with amylaceæ, 1; total, 14.

Amylaceous Food (not proprietary).—Alone, 6; with breast milk, 3; with milk, 5; with sterilized milk, 8; with pasteurized milk, 1; with peptonized milk, 1; total, 24. (9 of these were oat meal).

Table Food.—Nothing else mentioned, 11; with condensed milk, 1; total, 12.

Mellin's Food.—Nothing further stated, 42; with condensed milk, 22; with sterilized milk, 16; with pasteurized milk, 2; with other proprietary food, 1; total, 83.

Malted Milk.—Nothing further stated, 44; with cream, 1; with amylaceæ, 1; with other proprietary foods, 2; total, 48.

Condensed Milk.—Alone, 32; with milk, 1; with cream, 1; with other proprietary foods, 3; with table food, 1; total, 38.

Reed & Carnrick's Soluble Food.—13.

Imperial Granum.—6.

Liebig's Food.—Alone, 1; with condensed milk 1; total, 2.

Lactated Food.—Alone, 3; with condensed milk, 1; total, 4.

Nestle's Food.—Alone, 1; with sterilized peptonized milk, 1; total, 2.

Among other articles of diet mentioned by correspondents, each in one instance, are: Gardner's food, Robinson's barley, Ridge's food, Brush's food, animal broths, Bartlett's pepsinated food, Lactopræparata with Malted milk.

There are a number of instances in which the writers mention "proprietary foods" without further designation. In all 214 cases (60 per cent.) were fed on proprietary foods.

The effect of dietetic treatment has such an important bearing upon the etiological influence of diet that the whole matter will be discussed more fully under Treatment.

SYMPTOMS.—The symptoms in infantile scurvy are so typical and well known that they would appear to need little further

study. Nevertheless, the attention which has been directed to them by the questions of the circular has not been without fruit.

FIRST SYMPTOM TO DEVELOP AND ORDER AND TIME OF OTHER SYMPTOMS.—In response to this question the answers have not been altogether satisfactory. Undoubtedly in a large number such early symptoms as anæmia and mal-nutrition were overlooked or were not included by the readers as symptoms of the disease. Then in a large number, perhaps the majority of cases, answers have not made it quite clear whether the correspondent intended that a certain number of symptoms developed in the order in which the names are written, or whether they all were noticed at one time. Presuming that the first is the writer's intention, we make the following statement of the first symptom seen, basing this on 327 cases. The order of symptoms is too complicated and too uncertain to warrant a statistical arrangement.

FIRST SYMPTOMS SEEN.—Pain and tenderness, 145; affection of gums, 42; interference with motion, 36; anæmia, 27; cutaneous hemorrhages, 22; swellings, 16; restlessness, 6; anorexia, 5; debility, 5; diarrhœa, 5; constipation, 2; hemorrhage from nose, 1; hemorrhage from mouth, 1; hemorrhage from rectum, 1; hæmaturia, 3; "hæmatoma of tongue," 1; irritability, 3; vomiting, 1; fever, 1; opisthotonos, 1; sweating, 1.

PAIN ON MOTION OR HANDLING.—Pain is clearly a very prominent symptom of the disease. Generally it is evident only when the child is moved, or tries to move itself. Sometimes it is so intense that the approach of any one to the bedside is sufficient to cause the child to scream out through fear of being touched. Pain is reported present in 314 instances. In most of the remaining, no answer was made, and it is probable that the symptom could not have been a prominent one. The locality of the pain in the cases where there were accurate details was as follows:

Legs, 120; legs and arms, 25; legs and one arm, 11; legs and body, 4; one leg, 13; one leg and one arm, 1; one arm, 1; back, 1; back and legs, 1; back and leg, 1; back and thighs, 1; thighs, 1; hips and thigh, 1; one thigh, 2; one hip, 2; knees, 1; knees and ankles, 2; knees, ankles and shoulders, 1; knees, ankles and wrists, 2; knees and arms, 1; one knee, 1; one ankle, 1; ankles, 1; ankles and feet, 1; ankles and elbows, 1; elbow, 1.

PAIN WHEN AT REST.—In 91 cases pain seems to have been present, even when the child was still; while in 134 it is definitely stated as absent under this condition.

INTERFERENCE WITH MOTION.—The symptom variously described as *paralysis*, *pseudo-paralysis* and *disability* or *unwillingness to move* is reported frequently. It probably depends in every instance upon pain, since there is no evidence that actual paralysis occurs in the disease. In 319 cases interference with motion of this nature existed.

Rigidity is described as present in 96 cases and absent in 106. It is due to pain in most instances, but perhaps in others may have been occasioned or increased by the presence of swelling.

The parts of the body in which motion has been interfered with in any way in the cases reported, and the locality mentioned in detail, may be enumerated as follows:

Legs, 159; legs and arms, 55; legs and one arm, 14; legs and one hand, 2; one arm, 3; legs and thighs, 1; thighs, 3; one leg and one arm, 1; one leg, 27; one thigh, 2; hips, 1; one hip, 2; one hip and thigh, 1; one hip and knee, 3; hip, leg and shoulder, 1; hip, elbow and shoulder, 1; one knee, 4; one ankle, 2; ankles, knees, hand and wrist, 1.

POSITION OF THE LIMBS.—To a question regarding the position of the limbs, about which Barlowe speaks so definitely, there have been replies in 205 cases. In 17 of these the position was normal. In the balance we find the position of the limbs as follows:

Flexed, 152; extended, 23; flexed and abducted, 1; flexed and adducted, 1; flexed and everted, 1; abducted, 1; everted, 3; everted and extended, 2; toes extended, 1; feet extended, 3.

WEAKNESS OF THE BACK.—The occurrence of weakness of the back, a symptom which Barlowe says is marked, is mentioned as present in 97 of the cases reported to the committee and as absent in 108. In the remaining nothing is said of it.

DEPRESSION OF THE STERNUM.—This condition is likewise emphasized by Barlowe as being sometimes striking and characteristic. It is mentioned in 34 cases, but said to be absent in 170 others. It is not certain in the cases of the report how frequently the condition had developed acutely as a result of scurvy and how often it had already been produced by a previously existing rachitis.

SWELLINGS.—The effort has been made by analyzing the cases collected to determine the position of local swellings, whether these were situated in the joints or the shafts of the limbs, in the soft tissues or in the bones, and whether any redness was present. The answers are not clear in every in-

stance, and are frequently somewhat contradictory, partly, perhaps, from failure of the observer to understand the question, and partly from lack of careful discrimination between sub-periosteal and other effusions, and between effusion into a joint and that about it. The great irregularity also of the distribution of the swelling renders an accurate tabular arrangement too complicated. Remembering that in many cases more than one part of the body was involved and that the figures given do not mean that only the portion mentioned is affected in these cases, the following division may be made:

Joints (or probably oftener about joints) involved in 165 cases.

Location given in 101; *viz.*:

Knees, 73; ankles, 28; wrists, 12; hand, 1; elbow, 3; shoulder, 5; hip, 6.

Shafts of limbs involved in 179 cases. Location given in 123;

viz.:

Thighs, 59; legs (below knee), 61; "legs" (not further stated), 11; forearm, 5; upper arm, 4; "arm," 5; ribs, 1; scapula, 1; ilium, 1.

The gross results of the answers regarding the tissues in which the swelling occurred give:

Swelling in soft tissues, 97.

Swelling, sub-periosteal, 114.

Swelling in both situations, 16.

In 69 cases the swollen parts were reddened also. It is stated that there was no redness in 121. A more general swelling, to be classified rather as œdema, is described in 68 cases and stated to be absent in 98.

In regard to the swelling or protrusion of one or both eyes which has been described by writers, the symptom is said to have been absent in 110 cases and is reported present in 49. In 9 of these swelling only is mentioned, in 18 protrusion only, and in 22 both are referred to.

GUMS.—The condition of the gums and mouth is one of extreme interest. In 16 cases it is distinctly stated that the gums were entirely unaffected, while in 313 they were diseased. The degree of involvement varies from slight swelling to great sponginess and even ulceration. The degree and form of the affection in the cases suitable for study may be seen in the following table:

Swelling, absent, 14; present, 293.

Sponginess, absent, 27; present, 249.

Discoloration, absent, 23; present, 259.

Bleeding, absent, 64; present, 188.

Ulceration, absent, 101; present, 91.

The relation of the affection of the gums to the presence of teeth is of much interest. In nearly all the cases of scurvy in this report teeth were present, but what influence this has is not quite clear, since experience teaches that curiously it is usually the gums of the upper jaw which are most affected, although the lower teeth naturally are the first cut. Statistics on the portion of the gums involved were not furnished sufficiently to allow of conclusions; but regarding the teeth it is to be noted that of 359 cases suitable for comparison, teeth had already appeared in 314 instances, *i.e.*, 87.5 per cent.; while in only 45 cases, *i.e.*, 12.5 per cent., were there no teeth. In studying more carefully these 45 cases of scurvy without teeth, we may make the following analysis:

No teeth; gums normal, 21 cases.

No teeth; gums affected, 24 cases.

The conditions present in the latter group were as follows:

Swelling, 19 cases; sponginess, 14 cases; bleeding, 5 cases; discoloration, 17 cases; ulceration, 4 cases.

This is a proof that affection of the gums may occur equally well when there are no teeth as when teeth have developed. The fact that in the great majority of cases of infantile scurvy the presence of teeth and the affection of the gums is associated, depends merely on the fact that the disease generally develops at an age when teeth naturally have been cut.

CUTANEOUS HEMORRHAGES.—These have occurred with frequency in the cases reported. Accurate data are given in 353 cases. Of these, cutaneous hemorrhage is reported present in 182 and absent in 171. There is much doubt about the accuracy of the writers in their classification of the hemorrhages according to size, and the proper use by them of the descriptive names employed, inasmuch as the question on this point did not specify clearly. In 99 instances the presence of "ecchymoses" is mentioned. In 83 "purpuric eruption" is reported and in 37 "petechiæ." In 13 the nature of the lesion is not specified.

HEMORRHAGE FROM MUCOUS MEMBRANES.—Data are available in 361 cases. Of these there were no hemorrhages from any mucous membrane in 196, while in 164 they occurred. In 93 cases there was hemorrhage from the mouth. This includes the

cases where bleeding from the gums is described by writers. In 33 cases there was bleeding from the nose; in 2 from the stomach, and in 37 from the bowels. Cases of hæmaturia are not included here, and will be referred to later.

FRACTURES.—Fractures in infantile scurvy are usually separations of the epiphyses merely. Even this would seem to be rare, for fracture of any kind is mentioned in only 9 of our cases. In 342 it is distinctly stated to have been absent, and in the remaining the question is not answered.

FEVER.—Probably in the majority of the cases of the disease upon which this report is based no temperature record has been made. In 93 cases it is stated that there was no fever; in 182 it was present and in the remaining no answer is given. In the cases where present it is described as slight in 116 instances, moderate in 23, high in 8, and irregular in 6. Clearly, fever is not a prominent symptom of the disease, and probably often, when present, depends on accidental causes.

BOWEL MOVEMENTS.—The following conditions are mentioned:

Bowels regular, 74.

Bowels irregular, 15.

Constipation, 126.

Diarrhœa, 65.

Bloody diarrhœa, 12.

URINE.—Judging from the number of instances in which no answers have been returned, no examination of the urine has been made in most of the cases. It is reported as examined for albumin in 163 cases; in 33 of these albuminuria is reported and in 130 it was absent. Tube casts were present in 13 instances, absent in 13, and no observation reported in the others.

Properly speaking the occurrence of hæmaturia should be discussed under the title of hemorrhage. It is mentioned as present in 22 cases only. Of other abnormal conditions of the urine the following may be mentioned: Urine very acid, 1; urine scanty, 9; urine suppressed, 1; urine increased in quantity, 3; glycosuria, 1; hæmoglobinuria, 1; pus (from cystitis), 1; phosphates increased, 1; chlorides increased, 1.

ANÆMIA; MAL-NUTRITION.—These conditions, already referred to as often the earliest symptoms of infantile scurvy, may have been the first evidences of the disease in many of the cases on which this report is based. In other cases they must be regarded as complicating affections only. Answers are not full enough to allow of satisfactory conclusions on this point.

Anæmia is said to have been present in 254 cases, as follows:

Anæmia present (without specifying degree), 47.

Anæmia slight, 66.

Anæmia moderate, 32.

Anæmia marked, 109.

Blood examinations were made in 15 cases and the conditions noted as follows: The percentage of hæmoglobin was much reduced in all the cases, 8 in number, in which an examination was made, some being as low as 35 per cent. Of the 7 cases in which the red blood corpuscles were counted, all showed a reduction except 2. In these 2 the number was normal or nearly so, but the hæmoglobin was 50 and 35 per cent., respectively. Leucocytosis was present in 5 cases. Poikilocytosis in 2. In only one instance was there a differential count of the leucocytes made.

Of 217 cases in which the question is answered, *emaciation* is recorded in 167 and is said to have been absent in 50.

Mal-nutrition was observed in 178 cases out of 216, in which replies were made as follows:

Mal-nutrition present (without specifying degree), 108; slight, 20; moderate, 7; marked, 43.

RICKETS.—Infantile scurvy has so often been described as “scurvy rickets” and “acute rickets,” that the investigation of the actual relationship of the two diseases was one of the matters to which the committee directed especial attention. The question upon the circular reads as follows: (a) “Any symptoms of rickets present? (b) Slight or well marked? (c) What relation in time of development did they bear to the scurvy?” Satisfactory answers were received in 340 cases; in 152 of these (45 per cent.), there were symptoms of rickets present, slight in 72, marked in 64, and the degree not mentioned in 16. In the remaining cases (55 per cent.), rickets is definitely stated to have been absent. With regard to the relation in time of development, it is stated in 50 cases that the rickets was first present; in 14 that it developed with the scurvy, and in 2 after it. There does not seem to be evidence as far as this investigation teaches that the association of rickets and scurvy is at all intimate. Very possibly the same defect in diet which produced the one produced the other also, but the rapid recovery under treatment which the scurvy underwent did not apply to the rickets. This seems to indicate only accidental association of the two diseases; certainly not any causal relation between them.

OTHER COMPLICATING CONDITIONS.—A variety of affections are mentioned complicating scurvy in a number of cases, as follows:

Bronchitis, 5; cretinism, 1; enlargement of inguinal glands, 1; "cerebral symptoms," 1; convulsions, 1; pneumonia, 2; boils, 1; irritability, 1; vomiting, 4; eczema, 2; enuresis, 1; sweating of the head, 1; tympanites, 1; caput medusæ, 1; diaphoresis, 1; pertussis, 2; insomnia, 1; anorexia, 2; post nasal discharge, 1; measles, 1; restlessness, 1; phimosis, 1; indigestion, 2; laryngismus stridulus, 1; cystitis, 1.

DIAGNOSIS.—The study of diagnosis has been only incidental, based upon the mistakes made before the disease was recognized in certain cases. The only disease for which infantile scurvy was repeatedly taken appears to have been rheumatism. In several instances the affection of the legs was supposed to be due to sarcoma. The apparent paralytic condition has also been the cause of error in some instances.

DURATION OF ILLNESS AND PROGNOSIS.—The disease is essentially chronic; its course terminating only on the institution of proper treatment. This seems to be proved by the answers contained in the circulars. To the question concerning the *duration of the disease before the case came under observation*, replies were received in 306 cases, of which the following analysis may be made:

DURATION OF DISEASE BEFORE TREATMENT WAS COMMENCED.

DAYS.	CASES.	WEEKS.	CASES.	MONTHS.	CASES.
2	3	1	15	1	18
3	3	2	33	2	42
5	2	3	58	3	31
8	1	4	22	4	14
9	1	5	8	5	4
10	9	6	29	6	7
24	1	7	6	7	2
		8	1	10	1
		10	3	12	1
				2½ yrs.	1

Intensely interesting in this connection are the replies to the next two questions: First. *Duration of illness after treatment was commenced*, and second, *Duration of treatment before marked improvement was noticed*. To the first question replies concerning 308 cases were received. Of course those fatal during the attack of scurvy are not included here nor those which passed from observation.

Still more striking are the answers to the second question, as

to the time when marked improvement was first noticed. There are 311 cases suitable for study in this category, excluding fatal cases and those passing from observation as before. The replies are often astonishing. Nothing is more striking than the speed with which these reports show a grave constitutional disease disappearing under proper treatment. There is certainly no disease for which a more specific treatment can be said to exist. The replies to the last two questions may be conveniently stated in the following tables:

DURATION OF TREATMENT BEFORE MARKED IMPROVEMENT
WAS NOTICED.

DAYS.	CASES.	WEEKS.	CASES.	MONTHS.	CASES.
1	19	1	47	1	6
2	58	2	27	2	4
3	46	3	8	3	4
4	26	4	1		
5	19	5	1		
6	1	6	1		
7	2				
8	2				
9	1				
10	7				
12	2				

Reported as prompt recovery, 13; at once, 15; immediate, 1.

DURATION OF TREATMENT BEFORE RECOVERY WAS COMPLETE.

DAYS.	CASES.	WEEKS.	CASES.	MONTHS.	CASES.
1	6	1	48	1	28
2	5	2	54	2	14
3	14	3	36	3	5
4	8	4	14	4	2
5	5	5	6	5	1
6	1	6	14	6	2
8	2	7	1	7	1
10	17			8	1
12	3			9	1
13	3				
15	2				
16	1				

Reported as immediate, 4; almost immediate, 9.

TREATMENT.—Not so much could be learned of the value of treatment as could be desired on account of the fact that in nearly all cases we have a combination of diet and of medicinal measures, including the use of fruit juices, and it is impossible to determine absolutely which was the active curative agent.

Taking the cases in which treatment was effectual and which are suitable for study, the results may be stated as follows:

I.	Cases recovering under treatment with drugs only (no change in diet),	-	-	-	-	-	0
II.	Cases recovering under the use of fruit juice alone (no change in diet),	-	-	-	-	-	3
III.	Cases recovering under the use of beef juice alone (no other change in diet),	-	-	-	-	-	2
IV.	Cases recovering under the use of beef juice and fruit juice combined, with or without drugs (no change in diet),	-	-	-	-	-	6
V.	Cases recovering under the combined effect of change of diet, often including beef juice, and the employment of fruit juice, with or without drugs,	-	-	-	-	-	257
VI.	Cases recovering under change of diet, often including beef juice, and use of drugs (no fruit juice).	-	-	-	-	-	20
VII.	Cases recovering under change of diet alone, often including beef juice (no fruit juice),	-	-	-	-	-	38

These last two may be properly considered together, since there is no evidence that any treatment with drugs has an appreciable effect upon the disease. So many of the reported cases were treated with drugs alone without result before the correct diagnosis was made and other treatment instituted, that this belief is amply justified. Combining, therefore, divisions VI. and VII., and comparing the statements of the writers regarding the diet employed during treatment and that employed when the scurvy developed, we may make the following table based upon 58 cases.

Again the committee would state that no claim is made that the recovery was the result of the change, but that it quotes merely the statements of the correspondents to the effect that recovery took place after the change was made.

VI. AND VII.—RECOVERY FOLLOWING CHANGE IN DIET ALONE, WITH OR WITHOUT DRUGS. (NO FRUIT JUICE EMPLOYED).

Mellin's food to milk and beef juice,	-	-	-	-	2
“ “ to raw milk and beef juice,	-	-	-	-	1
“ “ to modified milk,	-	-	-	-	4
“ “ to modified milk and beef juice,	-	-	-	-	2
“ “ to diet and beef juice,	-	-	-	-	1
“ “ and sterilized milk to beef juice and broths,	-	-	-	-	1
“ “ and sterilized milk to sterilized milk and beef juice,	-	-	-	-	2

Mellin's food and condensed milk to modified milk,	-	-	-	-	-	1
“ “ and condensed milk to raw milk,	-	-	-	-	-	2
“ “ and sarcopoptone to fresh milk and beef juice,	-	-	-	-	-	1
Condensed milk to fresh milk and beef juice,	-	-	-	-	-	1
“ “ to sterilized milk and diet,	-	-	-	-	-	1
“ “ to lactated food and raw milk,	-	-	-	-	-	1
“ “ to sterilized milk,	-	-	-	-	-	1
Malted milk to milk and diet,	-	-	-	-	-	1
“ “ to raw milk and beef juice,	-	-	-	-	-	1
“ “ and amylaceæ to modified pasteurized milk and beef juice,	-	-	-	-	-	1
Sterilized milk to diet and beef juice,	-	-	-	-	-	1
“ “ to fresh milk and beef juice,	-	-	-	-	-	2
“ “ to raw milk,	-	-	-	-	-	4
“ “ to diet,	-	-	-	-	-	6
“ “ to raw milk and beef juice,	-	-	-	-	-	1
“ “ to sterilized milk and beef juice,	-	-	-	-	-	1
“ “ peptonized to raw milk and beef juice,	-	-	-	-	-	1
“ “ to pasteurized milk and diet,	-	-	-	-	-	1
“ “ “ “ “ “ - - - - -	-	-	-	-	-	1
Pasteurized milk to raw milk,	-	-	-	-	-	2
“ “ to fresh milk and beef juice,	-	-	-	-	-	1
“ “ to sterilized milk and broths,	-	-	-	-	-	1
Raw milk to amylaceæ,	-	-	-	-	-	1
Breast milk to peptonized milk and broths,	-	-	-	-	-	1
“ “ to sterilized milk,	-	-	-	-	-	1
Lactated food to raw milk and beef juice,	-	-	-	-	-	1
Reed & Carnrick's soluble food to modified milk,	-	-	-	-	-	1
“ “ “ “ “ to baked potato,	-	-	-	-	-	1
“ “ “ “ “ to beef juice,	-	-	-	-	-	1
Imperial granum to raw milk and beef juice,	-	-	-	-	-	1
Patented food to diet,	-	-	-	-	-	1
Ridge's food to diet,	-	-	-	-	-	1
Diet (poor) to diet (better),	-	-	-	-	-	2
Bartlett's pepsinated food to fresh milk,	-	-	-	-	-	1

It must be noted with regard to this table and those following that the term "modified" milk is used very loosely by the reporters. Occasionally it is specified to be laboratory milk, but much oftener this is not the case, and we are unable to know whether the modification was done at home or not, and whether the milk was heated or not. Presumably it was pasteurized in

many instances. Where the term "fresh" milk is employed in the table, we have been unable to learn by additional correspondence whether "raw" milk is meant or whether only a change from proprietary food to cow's milk is intended. The term "diet" as employed in the tables either expresses the fact that a large and varied number of different forms of diet were tried, too complicated to be detailed, or else quotes merely the statement of the writers that a change of diet was made, the original food probably being abandoned entirely unless otherwise stated.

The following table shows the food employed in divisions I., II., III. and IV., in which the diet was the same (except sometimes for the addition of beef juice) while the scurvy was developing and while it was recovering.

RECOVERY FOLLOWING WITH NO CHANGE OF DIET DURING TREATMENT.

I.	Treatment with drugs only,	-	-	-	-	0
II.	Treatment with fruit juice only,	-	-	-	-	3
	Mellin's food (milk sterilized),	-	-	-	1	
	Sterilized milk,	-	-	-	2	
III.	Treatment with beef juice only,	-	-	-	-	2
	Sterilized milk,	-	-	-	1	
	Raw milk,	-	-	-	1	
IV.	Treatment with combined beef juice and fruit juice only,					6
	Sterilized milk,	-	-	-	2	
	Sterilized milk and broths,	-	-	-	2	
	" " " amylaceæ,	-	-	-	1	
	Table food,	-	-	-	1	

Division V. contains by far the largest number of cases, 257 in all. The changes in diet employed are much too complicated to be stated fully in a table. Moreover, they are of little value, since the treatment was such a composite one, *viz*: change of diet combined with the use of fruit juice in every case, and often of beef juice and of drugs as well. A few of the more striking classes of cases may be selected as follows:

V.—RECOVERY FOLLOWING CHANGE OF DIET COMBINED WITH FRUIT JUICE, WITH OR WITHOUT DRUGS.

Condensed milk to milk, variously treated,	-	-	-	23
Imperial granum to milk, variously treated,	-	-	-	7
Lactopræparata to raw milk,	-	-	-	2

Lactated food to milk,	-	-	-	-	-	1
Reed & Carnrick's soluble food to milk, variously treated,	-	-	-	-	-	3
Malted milk to milk, variously treated,	-	-	-	-	-	38
Mellin's food to milk, variously treated,	-	-	-	-	-	21
Mellin's food and condensed milk to milk, variously treated,	-	-	-	-	-	19
Sterilized milk to fresh (probably always raw) milk,	-	-	-	-	-	34
Sterilized milk to pasteurized milk,	-	-	-	-	-	4
Pasteurized milk to "fresh" or "raw" milk,	-	-	-	-	-	9
Pasteurized milk to sterilized milk,	-	-	-	-	-	1
Raw milk to sterilized milk,	-	-	-	-	-	1
Breast milk to cow's milk, variously treated,	-	-	-	-	-	6

The conclusions to be drawn from this combined study of etiology and of treatment seem justifiable only to the following extent:

(1) That the development of the disease follows in each case the prolonged employment of some diet unsuitable to the individual child, and that often a change of diet which at first thought would seem to be unsuitable may be followed by prompt recovery.

(2) That in spite of this fact regarding individual cases, the combined report of collected cases makes it probable that in these there were certain forms of diet which were particularly prone to be followed by the development of scurvy. First in point of numbers here are to be mentioned the various proprietary foods.

(3) In fine, that in general the cases reported seem to indicate that the farther a food is removed in character from the natural food of a child the more likely its use is to be followed by the development of scurvy.

FATAL CASES.—Twenty-nine of the 379 cases are reported to have died. In 2 of these, death seems to have been remote from the attack of scurvy. Of the remaining 27 the causes as enumerated by the reporters are as follows:

Exhaustion, 6; cerebral hemorrhage, 3; diarrhœa, 2; bronchitis, 2; vomiting (?) 1; convulsions, 1; pneumonia, 4; malnutrition, 1; pulmonary hemorrhage, 1; ulcer of stomach, 1; syncope and nephritis, 1; doubtful, 4.

It is difficult to determine in how many of these the scurvy itself could be held responsible for the death; probably in few if any.

AUTOPSIES.—There have been handed in to the committee

the reports of 6 autopsies in all, some of them only partial. The salient points of each may be enumerated as follows:

Case of Dr. A. Caillé. Child of nine months, ill about three months. Autopsy showed hemorrhagic spots on the pericardium and surface of the liver; sub-periosteal hemorrhage of the long bones.

Case of Dr. L. E. Holt. Child of twelve months; ill for two months. Autopsy showed separation of the lower epiphysis from the shaft of the left femur; extensive sub-periosteal hemorrhage of the left femur; sub-pleural hemorrhages; broncho-pneumonia.

Case of Dr. L. E. Holt. Child of thirteen months; ill about two months. Autopsy showed sub-periosteal hemorrhage and separation of the lower epiphysis of the left femur; hemorrhages into the muscles of the left thigh, swellings about the opposite knee and both ankles; knee-joints normal; minute sub-pleural hemorrhages; well marked exudative nephritis; minute hemorrhages on the surface of the liver.

Case of W. P. Northrup. (The first autopsy in the United States). Child of eighteen months; ill about one month. Autopsy showed sub-periosteal hemorrhage of both tibiæ and both femora; detachment of the lower epiphysis of the left femur and maceration of lower end of shaft; broncho-pneumonia of left lung; no rachitic or syphilitic changes on microscopical examination.

Case of Dr. L. Starr. Child of thirteen months; ill for three months. Autopsy showed "right leg from knee to ankle stuffed with a puffy mass replacing normal tissue. Separation of both bones one inch above ankle."

Case of Dr. C. W. Townsend. Child of ten months; ill three to four weeks. Autopsy showed bloody serum in pleural cavity; perforating ulcer of the stomach; tubercular (?) process in peritoneum.

In conclusion the committee would thank publicly their correspondents who have sent their reports of cases and who are enumerated below. They are also greatly indebted to Dr. Wm. Schleif of Philadelphia for valuable aid in analyzing the circulars received and tabulating the results.

[Signed],

J. P. CROZER GRIFFITH, M.D., Philadelphia, }
CHARLES G. JENNINGS, M.D., Detroit, } Committee.
JOHN LOVETT MORSE, M.D., Boston. }

MINORITY REPORT.

1. From a study of this report and from due consideration of other known facts, scurvy appears to be a chronic ptomaine poisoning due to the absorption of toxins.

2. It follows the prolonged use of improper food and abnormal intestinal fermentation is a predisposing factor.

3. Sterilizing, pasteurizing, or cooking of milk food is not *per se* responsible for the scurvy condition.

4. A change of food and the administration of fruit juice and treatment of any underlying cause is the rational therapeutic procedure in scurvy.

(Signed) AUGUSTUS CAILLÉ, M.D.

DISCUSSION.

DR. CHRISTOPHER.—Does this report contain any definite conclusions as to the nature of scurvy? I heard Dr. Griffith give part of one. If it does, it seems to me we should strike them out, for we certainly cannot agree upon them. I will move, then, that all parts of the report giving conclusions be stricken out and all parts relating to the collection of data be retained and as such be published. (Seconded). Now, does it contain any conclusions? It certainly seems to me bad policy for this Society ever to adopt conclusions of any pathological matter. Even in so elaborately established a report as that of the Anti-toxin Committee, it is bad policy, and it looks like establishing scientific truths by legislation. The truths are valuable and should be published as far as possible.

DR. GRIFFITH.—I have had several letters, one just before I came from home, from men who wanted to know the conclusions of the committee. If this is to be of any value it must have some conclusions, because nobody except those deeply interested in scurvy, will read through the report and the tables. We are not to determine as a committee or a Society what produces scurvy, but we are analyzing reports of physicians. If 300 physicians say in so many cases these children were improperly fed and one physician says there was no improper food, the conclusion is before you. We do not say diet is the cause, but we say from the reports of these physicians, diet seemed to be the cause. As in the long German articles, the conclusions are placed at the end. We cannot get out of the fact that the conclusions are there. I think we ought to sum up here and there the facts that the report indicates.

DR. NORTHRUP.—Why should we gather all these statistics and then put them out in a mass? It is like tabulating the work of a hospital without commenting upon what they have done. I

would be very sorry to see this work set aside. It would be like a record that appeared not long ago of 500 cases of rheumatism without any comments.

DR. MORSE.—I feel that the conclusions that the committee has drawn up are entirely justified. If anything, they are much milder than the figures seem to indicate. But any further conclusions would call for some expression of opinion from the committee that must be based on their personal experience and it seemed best to leave that out. I feel very strongly that the paper should not be published without the conclusions.

DR. JENNINGS.—I sincerely regret that my opportunity for examining the report has not been sufficient to express an opinion upon it. From the examination I made I think that the conclusions of the committee are fully justified. Still, it would have pleased me better to have been present at the full meeting of the committee to hear the various expressions and perhaps my individual opinions might have been modified in that way. But as they stand now, the conclusions very nearly express my conclusions of the results of the investigation.

DR. BOOKER.—I went over the conclusions of the committee. There were some things I did not approve of, but we came by various modifications to something of an agreement and I am satisfied to stand by that agreement. The time we had for reviewing the report was very short, but I am willing to stand by the conclusions we came to.

DR. CAILLÉ.—I think that those conclusions are entirely safe and I do not personally object to them, but I would like to make a very short minority report based upon the cases I have collected.

DR. FORCHHEIMER.—Would it not be better to have the committee meet again and try to come to some conclusion, so that the report can be received as a whole? I move that the report be referred back to the committee with instructions to report to-morrow.

Dr. Christopher withdrew his motion and the motion made by Dr. Forchheimer was carried.

On the following day Dr. Griffith presented the revised report. Preceding this Dr. Rowland G. Freeman opened a discussion on the subject: "Shall all milk used for infant feeding be heated for the purpose of killing germs? If so, at what temperature, and how long shall this temperature be continued."

General discussion upon the report of the committee and the paper of Dr. Freeman then followed.

DR. BOOKER.—I have not had an opportunity to study the report required for a subject of so great importance to form an opinion upon it, and I beg to be excused from subscribing to it.

DR. BUCKINGHAM.—It appears to me that this report in all its bearings is as important a matter as can come before the Society at any time. It is important it should be settled right. It will be a serious thing to have it settled wrong. It seems to me that having heard the evidence offered to us and the reports made to us to-day, that there is just this to be said: if the evidence is accepted there is only one conclusion that can possibly be drawn and that is, the sterilization of milk has to do with the production of scurvy. When Dr. Caillé presented his minority report it seemed to me that what he did was to proffer the evidence that has come from his personal knowledge and the knowledge of his personal friends rather than the observations of people that he knows very little about. There are just two conclusions that can be drawn: Either sterilization of milk produces scurvy or collective investigations are not a safe way of getting information. For my part I am willing to accept either or both, and do accept both conclusions. But I hope very much we shall not insist upon the public accepting a lot of investigations until we are all united in accepting them. For my own part, I believe sterilization produces scurvy and the only reason it does not do it oftener is because of the imperfect way milk is sterilized. A great deal of milk masquerades under the name sterilized milk that is not sterilized.

DR. BOOKER.—When the sterilization of milk was introduced it was heralded by the medical profession as one of the most important advances in medicine. After ten years' experience in this method I am still of the same opinion, that it is one of the greatest advances that has been made in infant feeding. It was not claimed in the beginning that sterilization improved the digestion of milk. That part of the question has attracted so much attention that we have lost sight of the real object of sterilizing milk, that is, the prevention of sickness. The most important diseases which we have to deal with among infants are the digestive disorders in the summer time. The sterilization of the milk offers more advantages in checking or preventing those diseases than any other method which has yet been offered. Now has sterilization of milk so far done anything towards diminishing the summer diarrhœas of infants? We have not had reliable statistics to justify us in coming to conclusions. In the first place, sterilized milk has been used heretofore largely among the well-to-do, who are able to give the infants advantages which may of themselves be sufficient to avoid the disease. Again, the methods used for the sterilization of milk have not been sufficient. I believe that disease has been diminished by sterilization of milk and I believe the infantile mortality has been markedly reduced in New York, if I understood the President's address correctly, in the last few years since the sterilization of milk has been largely used there. Whatever the results have been, there is a brighter outlook in the future if we can continue along this line.

There is a great deal to be done in educating people in the handling of milk. If we can continue educating the people in this way, I believe a great deal will be done towards preventing disease in infants. It is possible that sterilization of milk may injure its nutritive properties to a slight extent. The chemical combinations in milk are held very loosely, and heating may be sufficient to destroy these relations. Heating may also coagulate some of the valuable proteids and to this extent the nutritive properties of milk may be somewhat injured. But the injury done by this is far outweighed by the greater advantage offered in preventing disease. As to the temperature at which milk should be sterilized, much depends upon the object of the sterilization and who is doing the sterilizing.

The ordinary methods of sterilizing milk do not sterilize the milk. They do not destroy certain harmful germs, germs which may not be harmful to the child if introduced into its body, but which are injurious to the milk. These germs are destroyed at a low temperature, such as the bacillus lactis aerogenes. Where the hope is only to destroy these germs, and it is understood that this is the object of the sterilization, the low temperature of pasteurization would be preferable, provided the pasteurization were conducted by a person who understood it or had the valuable apparatus presented by Dr. Freeman. Unfortunately, this apparatus is expensive and cannot be introduced where it is most needed. If we turn the sterilization over to the family I believe it is better to resort to sterilization than pasteurization. My own experience has been larger with sterilization than with pasteurization, and I do not believe there is a very great difference in the disturbance of the digestive qualities of the milk and the nutritive qualities by the one over the other. If the milk is to be kept for any time after its sterilization before it is used, as for journeys, it should be thoroughly sterilized, and that means a sterilization of three to six consecutive days at a high temperature for very many hours.

I particularly want to put myself on record that I do not believe the sterilization of milk causes the disturbances that have been claimed for it. I believe that in all the cases if we could get to the true knowledge of the previous management of the milk we would find it is that which has caused the trouble and not the manner of heating. It may be that certain idiosyncracies of the baby may be of such a nature that the sterilization of the milk will cause injury to the baby, but that may occur with any milk. When we analyze milk and find it normal as far as we can judge from the analysis, it may disagree with the infant. The sterilization, I believe, has no other influence in producing disease. I do not believe it produces scurvy. I believe that is due to the handling of the milk before and afterwards. Very few of us who have sent in reports have made examinations of the handling of the milk. I do not, and I believe very few have

done so. That is one reason I am not willing to subscribe to the report. If we could sift all these cases I believe we would find some condition in the milk, outside of sterilization, as we do in Mellin's food, which is mentioned in a number of cases under scurvy. It is very seldom Mellin's food is used as a food; it is not intended as a food but is more intended as a sugar for sweetening the milk. It is a valuable addition in some cases when it is used in that way. In all the answers I have received very few have said that they used Mellin's food alone; it is largely used with milk. If we could find out the true condition of the food before the scurvy was produced we would not find that the food bears such a relation to the disease. The whole subject is too complex and too little understood.

DR. WINTERS.—Two cases of scurvy which I have seen have a good deal of bearing, I think, on the discussion. One was a child nine months old that was brought to the dispensary too late to be reported to this committee. It was the fifth child, the other four in the family being absolutely healthy. The family history was excellent. This child had extensive changes under the periosteum which extended from the knee down to the ankle. It had the well known blue line beneath the two front teeth. The child had been fed exclusively for six months on sterilized milk. The other case was a baby five months old, brought to my office in consultation. The father and mother were healthy. This child was fed on Walker-Gorden milk and had never had anything else from its birth. That milk had been under sterilization for forty minutes. Here we have two typical cases of scurvy in which the family history has no bearing, one in which there has been four healthy children, both fed on proper food, and the scurvy, it seems to me, was due to the way in which the food was treated. The food in both cases was proper in composition and it had never caused the slightest digestive disturbances. In the case of the baby fed upon Walker-Gorden milk, the food was continued, but it was directed that it should be heated to 150° for ten minutes. The baby recovered. I have seen in all some two or three dozen cases of scurvy. The one case I spoke of was the only one that I have seen in dispensary practice, although I see thousands of children every year. My own impressions are that if the methods of feeding go on until we gradually meet the tenement people, we will also find scurvy in tenement practice. Every case of scurvy, except this one, I have seen in consultation. I have never used anything but a modification of food except in two cases, one a case which I saw with Dr. Reed, where beef juice and fruit juice were used, but the case was fatal.

DR. MORSE.—It seems to me that in approaching a collective investigation of any subject, the first thing necessary is to disabuse our minds of any previous ideas we may have held or failed

to hold. Then we should analyze our figures and study the result which they show, and we should not modify our result either because it does not agree with our previous points or because it seems to involve some issues which it is not our province to consider.

In regard to the sterilization of milk it seems to me that we must feel that the cooking of milk to any extent must modify albuminoids, proteid substances, and have some effect on the emulsion of fat. We must also admit that bacteria in milk do harm and for that reason we should heat milk to a certain extent. We have to meet two dangers, and in each individual case we must settle it according to our best judgment. One danger in connection with sterilized milk is that the public has forgotten that there is anything else in connection with baby's food except cooking it.

DR. JACOBI.—I can be very brief, inasmuch as I might have said something similar to what Dr. Morse has said just now. I arise to make a motion that the report and the minority report, and the remarks of Dr. Booker and this whole discussion be printed as it stands. We owe that to those who have done the work and we owe it to ourselves. We should not expect that we can solve the question forever. We must not expect that any report nor anything that any of us say must be accepted by the whole world. It is only a contribution to what has been known before. It would be a pity if the material which has been worked up should be lost. Whoever has spoken last upon this subject feels then that the subject is settled and that there is nothing further to be said about it. That has been so from the beginning of time and it is so now. What we say here and do here and our valuable report should be published, and the medical public who are going to read it will have something new at all events, either a confirmation of their original views or the opposite.

The Chair decided that the motion of Dr. Jacobi was not necessary, since the publication would be made according to the laws of the Society.

DR. ADAMS.—I am very much of the opinion expressed by Dr. Morse. We have a right to express our individual views. The investigation was entered into in all honesty and all deductions should be drawn from the figures presented by the public at large just as in the investigation of anti-toxin. We have a practice in the medical press of giving the public the benefit of individual opinions and this should be an independent investigation and not clouded by individual opinions. I have made observations in sterilizing milk, and I have my own opinions about scurvy, but I should be governed very much by the valuable report of this committee.

Now as to the discussion of milk. I, with very many mem-

bers of this Society, took up very early the high sterilization of milk, and was very soon convinced of its impracticability in the vast majority of cases. Then I resorted to the low sterilization. I have tried both methods in the most adverse circumstances, that is in a hot city where the temperature is often 80° for days at a time. I have done this under the most favorable circumstances with a good corps of good assistants and I have found one method has yielded entirely satisfactory results. While we have cardinal principles to govern us in feeding infants, we must study each individual case and adapt the methods accordingly. I have sterilized the milk in the Foundling Hospital and in the Children's Hospital, and then I pasteurized the milk in both of them, and I got bad results in both. Then I pasteurized at 150° in both and have secured the best milk possible and in this way I have had the best results. In the past two months I have not had any deaths among sixty children. In private practice, in those cases in which I could get them upon a supply of milk from a dairy under the medical supervision of the Society of the District of Columbia, I did not resort to either method but to my own modification of the milk by siphoning off the cream or the upper part of the milk. In this way I have not since January seen a case of gastro-enteric disease in my own practice. So I think our own experience should guide us rather than that we should lay down that all milk should be sterilized or all pasteurized.

DR. CAILLÉ.—I wish to say one word in Dr. Freeman's favor. I was asked by the council to sit as confrère in this matter, but I declined. I think we are getting a little hysterical in the matter of temperature and we will settle down to a solid basis after a while. The antiseptic principle applied to infant food is one of the greatest advanced in the last half of this century. If the opinion should go out from this Society that sterilization is bad practice, it would be a large retrograde step. It would be all right if it were possible to have absolutely clean milk from the time it is received until it is consumed, but since this is impossible, I prefer the sterilizing process rather than pasteurization, because pasteurization is a double process, a heating and a cooling process, and is a complicated thing for the common people to do. For that reason I prefer the temperature of boiling water. In reply to what Dr. Buckingham has said I will only say that my conclusions in the scurvy discussion are based upon the study of all the 370 odd cases, and Dr. Buckingham's conclusions are not based upon those cases. He has simply heard the report read and has not studied it as I have. Therefore, I think he is the bolder man of the two in formulating his conclusions.

DR. JENNINGS.—I have nothing further to add. Dr. Morse expressed my views even better than I did, and I stand by that expression.

DR. GRIFFITH.—I want to say that Dr. Morse has expressed my views. We are disposed to reflect upon the observers we do not know and say that we do not know anything about them; we do not know whether their milk was properly prepared, etc. I have read through every report sent me, with the exception of the few last ones, which included principally Dr. Holt's batch which I thought it hardly worth while to review. You will be surprised to hear with what care those cases are reported by men we do not know. We say here "sterilization"; they say "sterilized at 212° for so long." It was impossible in the report to make all these classifications. At the same time the committee has taken particular pains not to vouch for the statements made. We do not know how many are accurate, of course, and so we only took the figures received, added them up and gave you the results. That only expresses the tabulation of these cases. In regard to Dr. Freeman's paper, I would like to express my own custom and belief about sterilization and pasteurization of milk. I do not believe that sterilization does the harm usually attributed to it. It has been the custom among many people for a long time to so-called scald the milk, which raised it to near the temperature of boiling water. I think we do find comparatively little harm from this. At the same time we should get as near as possible the mother's milk. The sterilized or pasteurized milk is simply a lesser evil. Any condition of this kind is an evil and the only question is whether the evil of omitting it is not greater than the evil of doing it. Certainly in my mind it is better to boil the milk thoroughly and long, if necessary, to insure clean milk. If you cannot do that I would prefer pasteurization, or better than that, raw milk.

DR. HOLT.—Most of us believe that at times pasteurization of the milk is absolutely necessary, especially among certain people at certain times; but here is a thing, from the report that may be dangerous and let us admit that. I have seen cases precisely like those reported in which there seemed to be no question about the preparation of the food, and the change from boiled milk or milk which was raw was the only step toward recovery. In my early cases I gave no fruit juice but only changed the food. It seems to me there are many things done in private practice that cannot be done outside. But that there is danger in the prolonged use of sterilized milk, it seems to me, is something we cannot escape if we believe these men have told the truth. It is a thing that will occur at times, and when a baby gets sore gums and sore legs we will know what is wrong.

DR. BOOKER.—I would like to say that I believe in sterilization of the milk as a preventive measure and not as a food for the whole year. The proper time for using it is from June to October. In the cooler seasons, when we have tolerably pure milk, the raw milk is the best.

DR. FREEMAN.—Dr. Booker referred to the difficulty of pasteurizing in poor practice where you cannot have the proper apparatus. It is perfectly practicable to pasteurize at 80° C. without any apparatus at all, if we will exert a little care, the milk being put on the stove and watched until a film forms on it. That is not true of diluted milk but of pure milk. That requires about fifteen or twenty minutes as a rule. It gives you some error perhaps in the way of over-heating rather than under-heating. In regard to Dr. Caillé's remark that one of the disadvantages is that you have to keep all milk cool afterwards, I think that is not true. In sterilization where pressure was used and the sterilization continued about twenty minutes, we found a growth of bacteria within a day.

ACTION OF THE SOCIETY.

On motion, the majority report was adopted by a vote of 18 to 1.

PHYSICIANS REPORTING CASES TO THE COMMITTEE.

S. E. Leard, Livingston, Mont.; C. B. Leonard, Providence; I. A. Abt, Chicago; C. B. Warner, Port Henry, N. Y.; E. Lackner, Chicago; J. W. Marcy, Merchantville, N. J.; M. P. Hollingsworth, Princeton, Ind.; H. P. Stebbings, Chicago; A. B. Rosenberry, Arbor Vitae, Wis.; W. H. Teller, Philadelphia; R. H. Carver, Providence; F. P. Emerson, Roxbury, Mass.; C. Mayfield, Washington, D. C.; E. A. Balloch, Washington, D. C.; W. C. Hollopeter, Philadelphia; J. M. Miller, Philadelphia; H. S. Kinne, Philadelphia; C. T. Dercum, Philadelphia; N. K. Noyes, Duxbury, Mass.; J. L. Ames, Boston, Mass.; D. Choate, Salem, Mass.; O. F. Rogers, Dorchester, Mass.; G. R. Miller, Hartford, Conn.; H. L. Taylor, New York; E. B. McGraw, Pittsburgh, Pa.; W. D. Booker, Baltimore, Md.; Wm. Osler, Baltimore; J. F. Herrick, Ottumwa, Iowa; P. J. Eaton, Pittsburgh; F. W. Garber, Muskegon, Mich.; H. D. White, Hopewell Junction, N. Y.; A. H. Burr, Chicago; P. R. Egan, Washington, D. C.; E. J. Beall, Fort Worth, Texas; J. A. Anderson, San Francisco, Cal.; J. C. Wise, Washington, D. C.; J. P. C. Griffith, Philadelphia; H. Williams, Boston, Mass.; F. M. Crandall, New York; C. Strueh, Chicago; A. H. Bogart, Brooklyn, N. Y.; D.

G. Wilcox, Buffalo, N. Y. ; R. B. Gilbert, Louisville, Ky. ; F. W. Goss, Boston, Mass. ; T. C. Fanning, Tarrytown, N. Y. ; C. W. Sharpless, Seattle, Wash. ; C. A. Dewey, Rochester, N. Y. ; J. L. Morse, Boston, Mass. ; L. Fischer, New York ; C. G. Kerley, New York ; C. S. Merriman, Kansas City, Mo. ; F. M. Conn, Seattle, Wash. ; S. L. McCurdy, Pittsburgh, Pa. ; G. S. DeGroot, Mendham, N. J. ; W. A. Northridge, Brooklyn, N. Y. ; M. A. Veeder, Lyons, N. Y. ; H. B. Sheffield, New York ; A. M. Jacobus, New York ; W. A. Tremaine, Providence, R. I. ; W. H. Mays, San Francisco, Cal. ; J. M. Dunham, Columbus, O. ; A. Caillé, New York ; H. L. Smith, Hudson, N. Y. ; H. L. Machell, Toronto, Can. ; R. Engelman, Chicago ; B. C. Rachford, Cincinnati, O. ; H. M. Sherman, San Francisco, Cal. ; E. L. Kloop, Philadelphia ; S. J. Hudkin, San Francisco, Cal. ; J. J. Thomas, Boston, Mass. ; Sarah Hobson, Chicago ; E. Rosenthal, Philadelphia ; J. R. Wellington, Washington, D. C. ; A. C. Wentz, Hanover, Pa. ; E. D. Chesebrok, Providence, R. I. ; L. M. Yale, New York ; D. J. Evans, Montreal, Can. ; W. H. Barr, Agricultural College, Miss. ; A. Hand, Jr., Philadelphia ; W. F. Cheney, San Francisco, Cal. ; L. Starr, Philadelphia ; A. J. Gillette, St. Paul, Minn. ; P. B. Craig, Jersey City, N. J. ; D. Lichty, Rockford, Ill. ; E. A. Chapoton, Detroit, Mich. ; G. S. McKeough, Chatham, Ont. ; G. Duffield, Detroit, Mich. ; J. T. Cook, Buffalo, N. Y. ; C. W. Townsend, Boston, Mass. ; W. A. Edwards, San Diego, Cal. ; W. L. Carr, N. Y. ; J. W. Cokenower, Des Moines, Ia. ; J. F. Pember, Janesville, Wis. ; G. L. Hogan, Helena, Mont. ; T. S. Roberts, Minneapolis, Minn. ; G. H. Whitcomb, Greenwich, N. Y. ; C. P. Putnam, Boston, Mass. ; H. A. Laurent, New York ; W. B. Warner, Red Bank, N. J. ; M. F. Hobart, Boston, Mass. ; M. Deschere, New York ; M. A. Starr, New York ; D. Bovaird, New York ; T. M. Rotch, Boston, Mass. ; E. E. Graham, Philadelphia ; J. F. McCormick, Jamaica Plains, Mass. ; B. H. Broadnax, Broadnax, La. ; A. Jacobi, New York ; R. T. Bullard, Los Angeles, Cal. ; J. H. Fruitnight, New York ; H. S. Stokes, New York ; F. E. Sondern, New York ; A. Seibert, New York ; H. N. Read, Brooklyn, N. Y. ; H. A. Fairbairn, Brooklyn, N. Y. ; P. Scott, Brooklyn, N. Y. ; R. F. Ives, Brooklyn, N. Y. ; G. W. Colby, Brooklyn, N. Y. ; I. M. Snow, Buffalo, N. Y. ; A. Matheson, Neillsville, Wis. ; R. B. Kimball, New York ; G. E. Brewer, New York ; M. H. Shep-

ard, Brooklyn, N. Y.; V. P. Gibney, New York; F. M. Lloyd, Brooklyn, N. Y.; L. E. Holt, New York; H. Gibney, New York; C. H. Richardson, New York; J. Leidy, Philadelphia; J. H. Musser, Philadelphia; E. P. Davis, Philadelphia; C. G. Jennings, Detroit, Mich.; T. S. Southworth, New York; A. S. Blackader, Montreal, Can.; R. Moyer, Ogden, Utah; W. P. Northrup, New York; T. H. Hay, Milwaukee, Wis.; J. G. De Veaux, New York.

SHOULD ALL MILK USED FOR INFANT FEEDING BE
HEATED FOR THE PURPOSE OF KILLING GERMS?
IF SO, AT WHAT TEMPERATURE AND HOW LONG
CONTINUED?

BY ROWLAND GODFREY FREEMAN, M.D.,

Pathologist to the Foundling Hospital; Pathologist to St. Mary's Free Hos-
pital for Children, New York.

Although in this paper I will present no new facts, I hope to be able to make my remarks interesting by presenting to you a summary of the answers I have received to the questions I sent to the members of the Society relating to the heating of milk for the destruction of germs. I wish also to express my thanks to the members of the Society for their very courteous co-operation, which has made it possible to present this summary of the opinions of many of the most prominent pediatricists of the country. The questions I sent out were rather hurriedly drawn up and were not perhaps as definite in their meaning as they should have been, but in general the answers received gave evidence that they were understood as was intended.

I have received 37 answers, of which 34 were categorical and 3 in the form of letters. One of these letters gave details of a process used by the writer in which the milk was twice boiled, while two letters gave no details and were thus not suitable for this purpose.

In answer to the first question, "Do you consider that milk is rendered more digestible by sterilization or pasteurization?" the answers were: Yes, 3; a qualified yes, 1; no, 19; a qualified no, 4; while three thought it less digestible when sterilized and four more digestible when pasteurized. The great majority then consider that heating does not render the milk more digestible.

In answer to the second question, "Is sterilization or pasteurization to be advised for this purpose?" the answers are as follows: Yes, 1; qualified yes, 1; no, 21; qualified no, 1; pasteurization, 5.

The third question, "Would you recommend that milk should always be heated for this purpose?" was answered as follows: Yes, 3; qualified yes, 4; no, 25; qualified no, 1. It is

thus evident that a very large proportion of these gentlemen believe that milk may be fed raw under certain conditions.

The fourth question, "If not always, when, if ever, is it imperative?" The answers may in general be grouped as follows: Always, 1; when not kept cold, or in summer, 17; in gastric or enteric disorders, 4; when dairy hygiene is questionable, 12; during epidemics of scarlet fever, measles, cholera, or typhoid fever, 1; when milk is old, 3; in cities, 7.

The fifth question, "Do you prefer pasteurization or sterilization?" brought the following answers: Pasteurization, 23; pasteurization under certain circumstances, 6; sterilization, 1. Including the letter mentioned before, we would have 2 preferring sterilization; one prefers pasteurization in private practice and sterilization in hospital work.

As to the temperature for pasteurization: 1, uses 140° for 15 minutes; 1, 150° for 30 minutes; 2, 155° for 30 minutes; 1, 155°-158° for 15 minutes; 3, 155°-163° for 15-30 minutes; 1, 160° for 20 minutes; 1, 160°-167° for 20-30 minutes; 1, 160°-170° for 35 minutes; 1, 160°-177° for 20-30 minutes; 1, 160°-170° for 35 minutes; 1, 165° for 35 minutes; 18, 167°, for intervals ranging from 6 to 35 minutes, the majority being from 20 to 30 minutes; 1, according to Walker-Gorden method.

The question concerning the duration of sterilization was answered by only 13 members, the time varying from 15 minutes to 1½ hours.

The last question, "Are there any practical disadvantages in heating milk for sterilization?" brought forth a variety of answers, as follows: Yes, 2; no, 2; less digestible, 7; sterilized milk less digestible, 3; less nourishing, 6; possibly a contributing factor in scurvy, 8; objectionable taste, 4; objectionable odor, 1; change in color of milk, 1; destroys emulsion, 1; cream not evenly mixed, 1; constipating 2; household sterilization unsatisfactory, 1.

These replies seem to show a remarkable unanimity of opinion of the members of the Society, throughout the country the predominating opinion being that raw milk would be the best food were it possible to obtain it clean, while a considerable number are evidently willing to take their chances with raw milk during certain seasons of the year and under certain conditions of dairy hygiene.

It was, therefore, surprising to me that more of the members had not made use of pasteurization at a temperature lower than 167° F. There seems ample evidence that 155° F. for 30 minutes (a temperature exposure which does not change the taste of milk) is sufficient, but only six of all the replies advocate this temperature. One answer, which coincides very nearly with my views, comes from Dr. Victor C. Vaughn of the University of Michigan, whose authority in such matters is widely recognized. This I will take the liberty to present in full:

“DEAR SIR:—I will answer your questions as follows:

1. I do not think that milk is rendered more digestible by sterilization or pasteurization.

2. Sterilization or pasteurization is not advised for the purpose of rendering milk more digestible.

3. If milk could be obtained from healthy animals under complete aseptic precautions, I do not think it would be necessary or desirable to have it heated before feeding it to children.

4. Practically, sterilization or pasteurization is imperative because milk is not obtained at all times from healthy cows, and very rarely, if ever, under aseptic precautions.

5. I prefer pasteurization to sterilization. Pasteurization should be carried out at a temperature of 155° to 158° F. When milk is heated to 160° F., it is so changed that a marked difference in taste is produced.

6. I think that a temperature of 155° to 158° F. maintained for fifteen minutes, is sufficiently active to kill toxicogenic germs that may be present, provided that the milk, after having been heated, be kept at a very low temperature. The keeping of milk at a low temperature after heating and before it is fed to the child, is, I think, absolutely necessary, because we know that even boiling does not destroy the spores of certain harmful germs in milk; but these spores do not develop at a low temperature, and there is reason for believing that these germs do not develop in the body.

7. If milk is sterilized, I think fifteen minutes long enough time.

8. There are practical disadvantages in heating milk for sterilization. Some of these practical disadvantages are inherent and others are accidental and avoidable.”

It does not seem to me that our dairy hygiene even under the best circumstances, has reached a point where it can produce a

raw milk which is an absolutely safe food. Cow's milk must be obtained by pressure on the teats of a cow, and these teats hang beneath an udder which is covered with hair, and from the belly of the cow which is also covered with the same hair-covered hide. Moreover, this portion of the cow is particularly liable to be soiled with dirt as it comes in contact with the ground when the cow lies down. Its hairy covering, moreover, holds the dirt, which is gradually shaken out by friction. If the cow has loose fecal movements these run down the inner surface of the thighs and the posterior portion of the udder. The contamination dries on the udder in the air, and during milking is apt to fall as dust into the pail. Moreover, the milk ducts of the cow may contain many bacteria, although usually contamination from this source is not great. In some cases, however, it is considerable, and then it is not always eliminated by throwing away the milk from the first few squeezes of the teat. I have found it present in the milk forced out by even the fortieth squeeze. Milk thus may become contaminated in the milk ducts, and at any rate has to be obtained from a bad immediate environment. Many efforts are being made to minimize these dangers, but with the best methods now used they still exist to a considerable extent.

But this is not all. The milkman's hands are almost never clean and of necessity hardly can be. The milkman must labor hard with his hands all day, causing a thick, rough callous which is difficult to clean, if an effort to clean them is made. His hands are employed in handling manure, and in attending to many duties involving contamination. Occasionally they are used during the day in waiting on some one sick with a contagious disease, and when such is the case, the consumers of the milk are apt to suffer. If wet milking is used, the milkman's hands are practically washed over the milk pail.

We have here again in the milkman a danger to the milk which cannot yet be eliminated, and even with great care on the part of dairy superintendent it will be difficult to entirely do away with these dangers.

Epidemics due to milk have originated from a mild unrecognized case of typhoid in a milkman, also from a beginning diphtheria in a milkman. Such sources of danger may exist in very carefully conducted dairies, although the liability to them is much diminished.

It has seemed to me worth while to go over these dangers to milk and to consider the difficulties in eliminating them in connection with the matter of heating milk, to show how difficult this problem of obtaining clean milk really is.

The original contamination of milk up to the time of bottling in well-conducted dairies where great efforts are made to obtain clean milk, rarely amounts to less than five thousand bacteria in each c.c. By the time such milk reaches the consumer, the contamination is still greater, and if, as is usual, the milk is used during the twenty-four hours following delivery, it is apt to be very considerable before a fresh supply arrives, and is probably as a rule something between fifty thousand and five millions a c.c., or is roughly between three thousand and three hundred thousand a drop. These bacteria are for the most part air bacteria, but they may be putrefactive bacteria, or toxin producing bacteria, or pathogenic bacteria, and thus may produce in the infant that is fed on the milk a gastro-enteritis or acute poisoning, or the infectious disease of which the special organism present is the cause. Many instances representing each of these classes of illnesses due to milk have been reported.

Does it seem right in view of what we know of the bacteriology of mother's milk to give such contaminated milk raw to infants?

It has seemed to me that although fresh uncooked milk is the ideal and rational form for infant feeding, the practical impossibility of obtaining cow's milk clean has rendered some form of sterilization necessary. It does not seem fair to put into an infant's stomach a food containing thousands of bacteria in each drop, these bacteria being of unknown quality and very possibly of dangerous and pathogenic nature.

For the present then some sort of sterilization, it seems to me, must be used. High temperature sterilization causes certain chemical changes in milk. The change in the taste of milk occurs at 70° C. (158° F.) and the changes found by chemists begin with a temperature of about 80° C. (176° F.) Clinically it has been observed that children fed on milk heated to a boiling temperature do not thrive as those fed on raw milk, and also that this bottled food seems to be a predisposing cause of scurvy. On this account then it would seem that the lowest temperature which is efficient should be used for sterilizing. Moreover, a lower temperature continued for a long time is as sufficient in its

bactericidal action as a much higher temperature for a very short period. We should then use a temperature for heating which is the lowest which, when continued for a considerable time, will destroy with certainty all those pathogenic bacteria most feared in milk, as well as the bulk of the bacteria present.

As I stated before this Society two years ago, I believe that on the considerations just mentioned, 68° C. (155° F.) for thirty minutes, followed by rapid cooling, is the best temperature exposure. Such a temperature will destroy the germs of diphtheria, typhoid fever and tuberculosis, and so many of the other germs present that a plate planted from milk so treated and kept at a laboratory temperature will usually show no growth in twenty-four hours. At the same time this milk has not been heated sufficiently to give it a "cooked milk" taste or to change its taste at all, and the temperature to which it has been exposed is more than ten degrees centigrade below that at which the chemical changes in milk due to heating are said to take place. It is, however, evident that pasteurization at this temperature has been as yet but little used. I would urge on those gentlemen who have used a temperature of about 167° F., and who are inclined to favor raw milk, to try first pasteurization at this lower temperature. In using this temperature, care must be exercised that approximately this temperature is sustained for half an hour, and equally that the milk is immediately and rapidly cooled and kept cool. Raw milk, I believe, cannot yet be considered a safe food.

205 WEST FIFTY-SEVENTH STREET.

THREE CASES OF AMAUROTIC FAMILY IDIOCY.

BY A. JACOBI, M.D., LL.D.,

New York.

Sigm. Freud (*Nothnagel Spec. Pathol. u. Therap.*, Vol. ix., 2d Part, 2d Division, Page 255), in a special chapter of his work on infantile cerebral paralysis, discusses the family and hereditary forms of the latter affection. They differ in this, that sometimes more than one case will make its appearance in a single generation; in other instances, subsequent generations may exhibit the very identical or similar morbid processes. He feels justified in proposing the following classification of all the cases which may come under this head:

1st. Cerebral paralysis may occur in more than one member of a family. The cases are identical or similar in different persons and stages. Strabismus or spastic hemiplegia may be met with.

2d. Typical diplegiæ are met with in the same family or in consecutive families.

3d. Such cases as may be observed under the same circumstances, and may be defined as typical diplegia, though they differ from it to a certain extent.

4th. Such family disorders of infancy as are markedly different from infantile cerebral paralysis.

5th. The family—or hereditary—affections of later life.

Feeble intellects, with progressive muscular atrophy, were met with by Hofmann in four brothers and sisters; atrophic bulbar paralysis has been observed in families, and under hereditary influences; relapses of spinal paralysis—after recovery was complete—have also been noticed. Then there are the various forms of Little's disease—that is, paralytic conditions dating from protracted labors. There is Friedreich's disease,

with tremor, oscillating gait, which is not influenced by closing the eyes, with nystagmus, and slow and monotonous articulation, but with no spasm, with extinction of patellar reflexes, with little disturbance of sensation, and with deformed foot and scoliotic spine; finally Marie's "hérédo-ataxie cérébelleuse," also (like the former) with tremor, oscillating gait, which is not influenced by closing the eyes, with nystagmus, and slow and monotonous articulation; but (to distinguish it from Friedreich's disease) with no deformities, increased reflexes, occasional contractions, and more disturbed sensation and affection of the eye muscles. All of these show the possible varieties, differences and degrees. In only a few the anatomical diagnosis can be made so correctly, as, for instance, in Friedreich's disease, which consists in a degeneration of the long spinal nerves and in smallness of the cord; or that of Marie's, in which the cerebellum is found to be reduced in size.

Under No. 2 Freud subsumes the cases of amaurotic family idiocy—a preliminary title given by Dr. B. Sachs, of New York, to a limited number of cases of a peculiar nature, which will be described hereafter. It appears to me that the classification of Dr. Freud is too indefinite to be accepted. If anything, it shows that the varieties, anomalies and alterations, both anatomical and clinical, in morbid conditions of the nervous system, which, result in or are complicated with idiocy, are too manifold, or, in many cases, too closely related to each other to permit of iron-clad boundary lines.

"Amaurotic family idiocy" is certainly exceptional in this respect, that it permits of a ready clinical diagnosis. Whether the anatomical lesions underlying all the cases, or their embryological development are identical, remains to be seen.

Dora R. was presented at my college clinic four months ago. Through a mistake of mine, or that of an assistant—certainly not through that of the gentleman who examined her eyes for him—I gained the impression that the retina showed either a plain bilateral hemorrhage or a bilateral glioma. The brief exhibition of the infant before my class, undertaken without further examination and without a history, was made for the sole purpose of presenting what I thought to be a fair illustration of Locke's dictum: *Nihil est in intellectu quod non antea fuerit in sensu*, "the road to intellect goes through the senses." It appeared to me that the defective intellectual development of the

child might be due to her increasing blindness and to the corresponding lack of sensory food. When, however, the defective muscular innervation was noticed, and when Dr. Gruening saw the child and corrected my erroneous opinion in regard to the condition of the retina, the case looked different.

She was one year old, and the fourth child of healthy, not neurotic, not syphilitic, not alcoholic, Semitic parents. No hereditary taint known. Their first child was six, the second four, the third three years old; all were in perfect health. Labor was easy; no asphyxia; no convulsion. Infant was nursed by her mother until the present time, with the exception of additional meat-soup and oatmeal decoctions during the last month; was considered normal by the mother during the first few months; she did not remember, however, when the baby smiled the first time. The first tooth, a lower incisor, appeared at ten months; three upper came since; four more teeth were protruding. The fontanelle was about to close; no pulsation was felt through it. Circumference of the head, $40\frac{1}{2}$ centim.; forehead rather narrow.

The infant never was able to sit alone or to hold up her head. When she was seven months old she did not notice so much or play so well as formerly; gave but little attention, cried but seldom, and slept a great deal. Some months ago spastic contraction of the upper extremities was observed repeatedly. Mother believed that during these last three months the infant noticed more, again. She felt certain that her eyes followed a light occasionally, and, at times, persons.

But her intellect was feeble when presented; she did not appear to notice, and she did not smile, except occasionally, in a listless manner. There were rachitical curvatures with convexity outwards; the epiphyses of the radius and ulna were rather enlarged, but other symptoms of rickets were absent. There was no Harrison's groove, there was no excessive perspiration, and no falling out of hair on the occiput. The fontanelle has been mentioned as nearly closed, rather earlier than usual; but there was no hyperostosis nor anything pointing to premature ossification; no constipation. I mention the latter circumstance because the involuntary muscles, so often and badly affected in rachitis, did not suffer here like the voluntary. These, all over the body, were flabby and inactive. Her motions were listless; her head found no muscular support; muscular

contraction under the influence of electricity was insufficient. The response to faradic irritation was diminished and quite slow; a rather strong current exhibited a contraction after from one to two seconds only. Galvanic irritability was also diminished, to a certain extent the lower extremities, much more so the upper; decided reaction of degeneration. Patella reflexes were rather normal, perhaps a little exaggerated; no ankle clonus. There was some slight dulness (normal) over the manubrium sterni. Thyroid seemed normal in extent and consistency. Pupils did not, or but very slightly, respond to light, either direct or reflected; no nystagmus, no strabismus, no convulsions; heart, lungs and kidneys were normal. The shape and consistency of the eye were normal. There was a white patch with a red centre, instead of the macula lutea and beyond it; in its centre was a depressed spot. The optic disk was pale and gray.

This degeneration is the same which has been described in all the cases hitherto observed, the first time by Waren Tay and Kingdon (in the former's first cases) as early as 1881.* The fundus was found normal until after the third month; it was then that the first haziness began to show itself in the macular region. The condition became worse about the fifth and sixth months, while vision grew worse gradually, until total blindness set in.

SECOND CASE.—Lina K. was presented in my office on the 23d day of September, 1897. Father and mother were each twenty-six years of age, both of the Semitic race, and emigrated from Russian Poland. They had another girl that died at the age of one-and-a-half years, two years previously. She never was strong enough to sit up or to hold her head. She was nursed until she was fourteen months old. At that time she had her first tooth; four when she died. She had her first convulsion with nine months. During the attack she appeared to be breathless; the eyes were turned up; other spasms occurred several times a day, both in the upper and lower extremities until she died. The face began to participate in these

* Since the subject has been discussed, amongst others, by Goldzieher Magnus and Knapp in 1885; by Wadsworth in 1887; by Sachs in 1887, 1896 and 1898; by Hirschberg in 1888; by Kingdon in 1892; by Carter in 1894, and lately by E. C. Kingdon and J. S. Risien Russell in the (1897) eightieth volume of the *Med. Chir. Trans.* ("Infantile Cerebral Degeneration with Symmetrical Changes at the Macula").

attacks when she was fifteen months old. When Lina K., was first presented, there were no very prominent symptoms, except listlessness and muscular weakness; no convulsions. The eyes certainly followed the watch. The erroneous diagnosis of rhachitical feebleness, "pseudo-paralysis," was made, and elixir of phosphorus and the syrup of iodide of iron were ordered. On February 23d she was next seen in my service at the Vanderbilt Clinic when fourteen months old. She still appeared to follow the watch, sometimes listlessly; her hearing was good; the repeater near her ear made her start, and dilated the rather large pupils. The eyes were mostly turned upwards; the head hung back; muscular action was very slow everywhere; she never held anything in her hands. The bowels were costive, and moved hard and sluggishly only once in two days, after enemata. The left leg required a continuous current of 3, the right of $7\frac{1}{2}$ milliampères for muscular contraction; the interrupted current had to be quite strong to accomplish that. There was no reaction of degeneration. For several months past she had convulsions in both arms, hands, legs and feet; convulsions and apnoea appeared to come together with pallor; the attack lasted "a few minutes," and occurred first but three or four times a week, but lately as many times during the day; also in the night. Some were accompanied with laryngismus stridulus; if they were tonic, they were followed by a tearless cry. The mother said the infant smiled when talked to, an assertion which could not be verified. Respiration was shallow, pulse 132 and regular. Fontanelle was still open. When the mouth was opened, its right angle was slightly drawn up. The characteristic change of the retina and macula lutea were looked after, and found by Dr. J. Herbert Claiborne.

A brother of the above patient was, at my request, sent to me by Dr. John Huber. He was then four weeks old, May 28, 1898. Dr. R. O. Born examined the eyes for me on the same day, and reported them to be normal, so far.

THIRD CASE.—G., female, was sent by Dr. Michaelis on April 19, 1898. She was born July 30th, 1897, after a normal labor, and with only a moment's "suspended animation." She was breast-fed. Two months ago, so the mother reported, the infant could not yet sit up. The only apparent disturbance of her health was a bronchitis from which she had suffered six weeks ago. Reflexes were normal; pupils of normal infant size (rather large)

and equal; intellect and muscles certainly feeble. The parents—Semitic—had been married seven years before this first child was born. No miscarriages, no syphilis, some tuberculosis in the father's family. Dr. R. O. Born, whom I requested to examine the eyes for me, reported the presence of the characteristic change in the macula lutea, with no atrophy of the fundus as yet.

While these cases are presented, they are not expected to be utilized for a lengthy review of the subject; for there is a very concise story of what is known of the subject by Dr. Sachs in the third number of the *D. Med. Woch.* of this year. Inclusive of Waren Tay's first three cases, all occurring in the same family, and terminating fatally before the end of the second year, altogether twenty-seven have been published. In all of them there were the same symmetrical changes in the macula lutea, *viz.*: the white, somewhat spherical spot, with a brownish red centre, similar to what is seen in embolism of the central artery, with gradual atrophy of the papillæ, which, in 1885, were studied in our country by Knapp and by Wadsworth; the same lack of innervation as shown by weakness of intellect and of muscles, the same connection first pointed out by Kingdon (*Trans. Ophth. So.*, xii.) of the intellectual deficiency with the characteristic eye changes, and the slow but sure increase of the symptoms. It matters little, and depends greatly on the individual powers or opportunities to observe the progress of such a case, whether there is a little more or less rolling of the eye, or increasing indolence, or reflex anomalies. The general progress and the end have been the same, *viz.*: the blindness and idiocy became complete, nutrition was impaired (marasmus is often mentioned), and a fatal termination completed the history of the cases before the end of the second year. Dr. B. Sachs observed a single case up to the sixth year. In another single case Koller found the eye symptoms to be rather late compared with the intellectual defects, and Higier the retinal atrophy more pronounced than the spot on the macula lutea.

In one of the three cases described by Kingdon and Russell in the eightieth volume of the *Med. Chir. Trans.* of 1897 there was a marked adductor spasm of the lower extremities; in another various distortions by the overaction of muscles, and overextension at the right knee. Such variations—even the oc-

casional aural disturbances from hyperacusis to dull hearing—establish no essential difference in the general symptomatology.*

Our anatomical knowledge of the pathological changes underlying these uniform vital changes left, until a short time ago, and still leaves, much to be desired. In one of the four older autopsies the arachnoid and pia were found thickened, the cortex hard, the cerebral fissures strongly marked, the sulcus Rolando and the fissure of Sylvius confluent (always a low form of development), and the island of Reil uncovered. The pyramid cells were but rarely normal, both lateral columns have been found degenerated. In one of the three cases lately described by Kingdon and Russell there was an extensive apparent sclerosis of the pyramidal tracts, and in the pons, the medulla, and the spinal cord. They suggest that possibly many of the pyramidal fibres may never been myelinated.† Moreover the anomalies of the brain and the cord are not contiguous; indeed just as little so, as for instance, in a disseminated sclerosis in which pons and the upper part of the cord may be quite normal, while there are similar and cotemporaneous changes in the cerebrum above and the cord below. In H. Higier's case (quoted from *Deutsche Z. f. Nervenheilk.*, x., 1897), which occurred in a family in which two infants had previously died from probably the same condition, there was thickening of the retina in the area of the macula, excavation of the papillæ and atrophy of the optic nerve fibres. There was no vascular disease or inflammatory process; but atrophy of the cortical pyramid cells, descending degeneration of the whole pyramidal tract, and degeneration of the motor root of the trifacial nerve and of the pedunculi cerebelli.

The chiasma was found normal. The blood-vessels were also found normal, and there were no evidences, we are told, of anything like an "inflammation," at least, such as could be traced to the blood-vessels.

Now, what is the morbid process underlying the progressive, intellectual and physical change and decline? A mere arrest of development in early embryonal life, which has not been

* The internal ear does not suffer, or seldom suffers, in this disease. It is developed from the outer ectoderm, while both retina and optic nerve are, like the brain, from the medullary tube of the ectoderm.

† In all of their cases the cerebellum presented no evidence of degeneration or other change in the cells of Purkinje or those of the corpus dentatum.

proven, but presumed to exist by Dr. Sachs, cannot cause a *progressive* change such as is illustrated by the increase of idiocy and paralysis, and of the visible gradual alterations of the retina in a child that at first appeared normal.

The optic tissue consists primarily of radiated spindle-shaped cells which resemble the spindle-shaped cells primarily found in the early stages of the brain.

The suggestion is justified that the normal and the abnormal development of these spindle-shaped cells may go on *pari passu*, both in the embryonal eye and in the embryonal brain. Embryologists and histologists will perhaps elucidate the connection existing between the degenerative processes—which possibly is of an inflammatory nature originally—in cases like ours, and the accompanying ocular cerebral and spinal defects. To speak indiscriminately of a degenerative process pure and simple, without inflammation, either acute or chronic, is a questionable procedure as long as we are not certain or unanimous as to the essential requirements or character of “inflammation.” Moreover it should be remembered, that in several instances, sclerotic processes, frequently the results of known inflammatory processes, are claimed for the cerebellum and other parts.

In connection with the definition of “inflammation,” I suggest the following extracts from two sources: We read in Delafield and Prudden, a hand-book of pathological anatomy and histology, 5th Ed., 1896, p. 107: “The phenomena which are embraced under the name of inflammation are: degeneration and death of tissue; changes in the circulation of the blood; escape of the elements of the blood from the vessels; formation of new cells and new tissue. *These morbid changes either occur separately or are combined* in various ways. The growth of the body of pathogenic micro-organisms and the formation by them of toxic substances is a frequent inciting cause of inflammation;” and in *E. Ziegler Lehrb d. Allg. Pathol u. d. Pathol. Anatomie*, 9th Ed., 1898, I., p. 322, as follows (translated): “Inflammation may be caused by mechanical, thermic, electric or chemical influences, or by parasites. All these noxæ have this in common, that they produce a *local degeneration of tissue*, which—*when it is extensive or intense—is combined* with disorders of circulation and of vascular secretion.” (Italics mine—A. J.)

The latter, therefore, should not be expected in every form or case of “inflammation,” and to speak of degeneration in contra-

distinction to inflammation is rather a hazardous procedure in many instances.

The causes of an inflammation are not specific at all. Any noxa or injury may produce it, provided it be sufficiently intense to cause both degeneration of tissue and certain disorders of circulation, without producing complete interruption of circulation and necrosis of the tissues. The rapid physiological evolution of the spindle-shaped cells composing at that early period, both the optic tissue and the cerebrum furnishes the possibility of pathological alterations. Developmental over-activity in the embryo and fœtus may be looked upon from the same point of view as post-natal functional over-activity. The pathological anatomy of the newly-born heart or of the rachitic bone or of the over-exerted muscle of young or old prove the close proximity of physiological and pathological conditions. That is why I cannot divest my mind from supposing that the degeneration which has been described as common to the few autopsies of such cases as ours was originally of an inflammatory character. Though it should be remembered, that there may be extensive cell degenerations without *marked* disorders of circulation, resulting for instance from the influence of toxins.

The thickening of arachnoid and pia and the hardness of the cerebral substances noted in one of the autopsies hitherto made, appears to point in that direction. It appears not unreasonable to suppose that the same inflammatory (proliferative, hyperplastic and by and by perhaps finally cicatrizing) process takes place in the macula lutea, or what is to *become* macula lutea.* Its late development would explain why (while undoubtedly the anatomical conditions of idiocy may be nearly developed at birth, the alterations of the macula lutea are discovered late;

* Macula lutea is absent from the embryonal retina; even from that of the newly-born. According to Huschke and other older authors it is a remnant of the ocular fissure; in the opinion of many the fovea centralis represents the upper remnant of the retinal fissure. Würzburg states that the formation of the macula lutea is preceded by a duplicature—a fold of the retina; still, these duplicatures are met with in several parts of the retina. At the very spot where the fibres of the optic nerves enter in later stages, the interior of the eye, there is, according to Bergmeister, a layer of cylindrical cells during embryonal life. Here the edges of the coloboma are turned up and rarefied, while in the rest of the eye the margins join one another. In this way the physiological excavation is formed.

S. L. Schenck, *Lehrbuch der Embryologie des Menschen and der Wirbelthiere*, Wien, 1896, 2d Ed., p. 187.

why they gradually get more pronounced and more destructive; and why they need not always—as instanced in Koller's case—appear at a regulation time.

The child whose case was discussed before the annual meeting of the American Neurological Association of 1898, by Dr. F. Peterson, was born of a German mother twenty-eight years old, the father being a Russian, twenty-nine years old, both of the Semitic race. Their first child is seven years old and normal, the second is an idiot of five and one-half years of age that became blind at six months, the third is a normal child of four years, the fourth was observed to be blind when four weeks old and died idiotic at ten months of age, the fifth died at the Randall's Island institution when seven months and twenty days old with the symptoms of amaurotic idiocy. It was said to have played and laughed up to the third or fourth month. There were measles at five months, followed by diarrhœa; the media of the eyes were clear, the pupils equal and somewhat dilated (normal?). There was no tremor, but gradually a rotatory and up and down ocular spasm was noticed, the neck and knees became rigid, the surface exhibited some purpuric spots, and the child died. The autopsy was made by Drs. McAlpin and Ewing. The lungs showed broncho-pneumonia; the kidneys were congested; the other viscera were all normal; the liver showed some fatty infiltration; the brain weighed 22 ounces; its surface was œdematous. The condition of the two halves was identical. The cortex was deficient in cells, all of them were of minute size and moderately deficient in chromatic substance. The same condition was noticed in the motor areas. In the cervical cord the cells of the anterior horns were normal, below this normal region they were less in number and shrunken; the cells of the olives normal and abundant, of the corpora quadrigemina and geniculata less and small, those of the sympathetic ganglia of the aorta of the spinal tract, and of the optic nerve and chiasma, normal; and the connective tissue rather deficient in the brain, but not so in the spinal cord. While in the normal infant brain the cells are arranged in distinct vertebral and longitudinal columns, almost all the vertebral columns were missing, while the longitudinal rows were indistinct. Dr. James Ewing characterized the main features of

the specimen as small size and small number of cells, and increased number of blood-vessels.

In the discussion following the report of this case Dr. Ward O. Holden who had examined the eyes, stated that vacuolation was found in the ganglion cells of the eyes, but the nuclei were not granular and the nucleoli were distinct while Nissl's granules were present in the cell bodies. There were no thorough changes. Moreover he stated that the results of the autopsy might be incorrect because it had been made forty hours after death. As an instance of the import of *post-mortem* changes he related that bacteria coli were found in the blood-vessels, and from his remarks it seemed as if the diagnosis of amaurotic family idiocy might be doubted.

In the same meeting Dr. Wm. Hirsch detailed a case, the history of which was accompanied with extensive and elaborate demonstrations. There is no report which excels it in completeness and accuracy.* L. P. was seen first in July, 1896, when ten months old. It developed fairly until it was six months old. The mother, who lost two children when they were eighteen and twenty months old, respectively, reported that the child then became weaker and the muscles flabby. At that time there was some perception of light; reflexes were good, viscera normal, and the ophthalmoscopic appearance like that described above. It died July 17, 1897, and the autopsy was made four hours after death. The dura was adhering, the pia normal; there was no fluid and no œdema; no macroscopic anomaly, except a prolongation of the second temporal fissure.

Specimens were hardened in formalin 10 per cent.; one eye in formalin, one in Müller's fluid, and sections made in paraffine. Methylene, eosin, or hæmatoxylin were employed for staining purposes. The pyramidal tracts appeared degenerated as others observed them. In the cells of the anterior and posterior columns the nuclei were well defined, also the nucleoli; both of them were in many instances found near the edges of the cells. The cells were large, that is why they appeared too numerous. The motor cells were of double size, some apparently without nuclei, an impression mostly due to obliquity of section. Very few axis

* Dr. Hirsch gave me his manuscript to read, and demonstrated to me in his laboratory many of his charts and microscopical specimens, and kindly gave me some characteristic ones for presentation to the American Pediatric Society. His own complete report will undoubtedly appear soon.

cylinders and dendrites. Pericellular spaces small or absent. The nuclei surrounded by a dark zone. Nissl bodies disappear entirely in a granular mass. The light area contains a fine network; few normal chromatic bodies; medulla oblongata is found in the same condition, the changes in nuclei and cells are as above; in the olivary bodies the nuclei are markedly round; in the reticulated they are oval-shaped and pushed to the extreme end of the cells. In the cells of the trifacial nerve the nucleoli are hardly recognized. In the pericellular lymph spaces there are masses of detritus. Subcortical ganglia, thalamus and cortex are in the same condition. The pyramidal cells are changed and swollen, with displaced nuclei; also Purkinje's cells of the cerebellum.

The points Dr. Hirsch mainly emphasizes in connection with our subject are these: All the nerve cells, as far as examined, are changed in a peculiar way. There is chromatolysis and other degeneration. The cell bodies are large. The nuclei are displaced sideways. Neuroglia and blood-vessels are normal. Differences in the accounts of previous observers are explainable by insufficient methods of hardening or staining; Müller's fluid, for instance, shows the enlargement of the body of the cell but not its finer structure. The observed changes are pretty universal; there is neither an arrest of development nor a localization in the cortex, but as Kingdon and Russell assume, an acquired degeneration.

Where is its cause? As the infant is in good condition at first and the macula normal, the cells should be considered normal. No part of the embryonal history explains the future illness, which must be acquired. As the blood-vessels are found normal the process is not inflammatory(?). No systemic disease explains all the cell changes; there is no atrophy. That is why Dr. Hirsch believes that the only possible explanation is furnished by the presence of a toxic condition, which would correspond with experimental poisoning, the effects of which on the nerve cells, and on general nutrition, are different from all other disorders. What is the nature of the toxic substance? Perhaps the examination of the mother's and the infant's blood, and that of the mother's milk, will lead to the solution of the problem. At all events, Dr. Hirsch feels like weaning such infants, or refusing the mother's breast to any baby whose family history prevents cases of the same condition.

In connection with Dr. Hirsch's paper the characteristic appearance of the macula was made the subject of discussion by Dr. Ward O. Holden. He alluded to Treacher Collins, who explained the red spots by the thinness of the retina in the fovea centralis, while round about there was, in his opinion (which, however, he doubted himself), an intense œdema. This alleged œdema is, however, found in the healthy eyes of dead persons, and may result from the following of the retina round the macula in that condition. The real cause of the red spot surrounded by white discoloration is, according to Dr. Holden, as follows: In the periphery of the retina where the cells are scattered, they are large. In the macular region they are several (6-7) layers deep and mostly small. In the peculiar degenerative process of amaurotic family idiocy these cells are of increased (double) size, and their contents dense and turbid. Hence the white discoloration. The fovea centralis has no layer of ganglion cells—hence the choroid shows through.

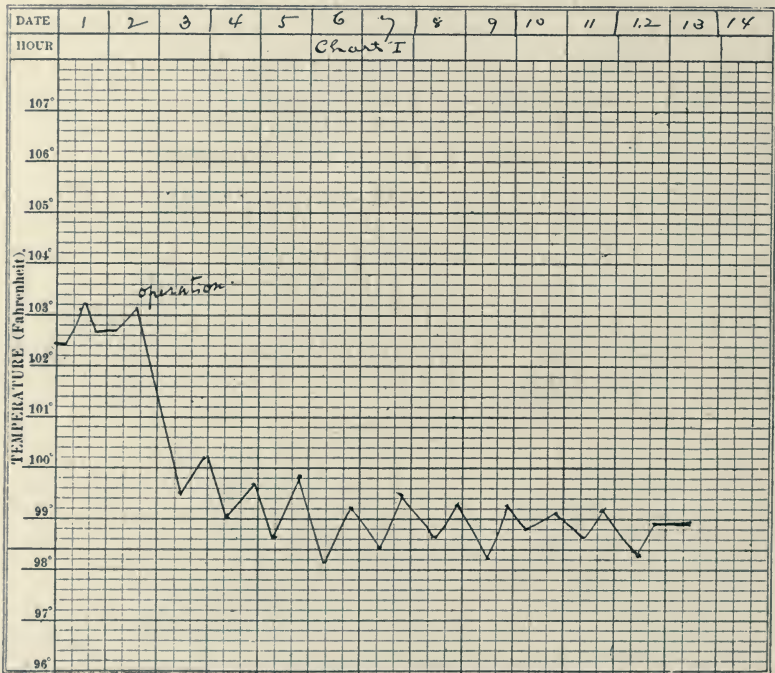
The atrophy of the optic nerve is always observed long after the changes in the macula lutea. It develops very gradually. According to Koller, the behavior of the children with regard to their vision was not normal at a time when no ophthalmoscopic changes were visible, so that the conclusion is permissible that vision had been impaired by cerebral degeneration. (*New York Medical Record*, July 9, 1898.)

THE SIGNIFICANCE OF FEVER FOLLOWING OPERATIONS FOR PYOTHORAX.

BY AUGUSTUS CAILLÉ, M.D.,

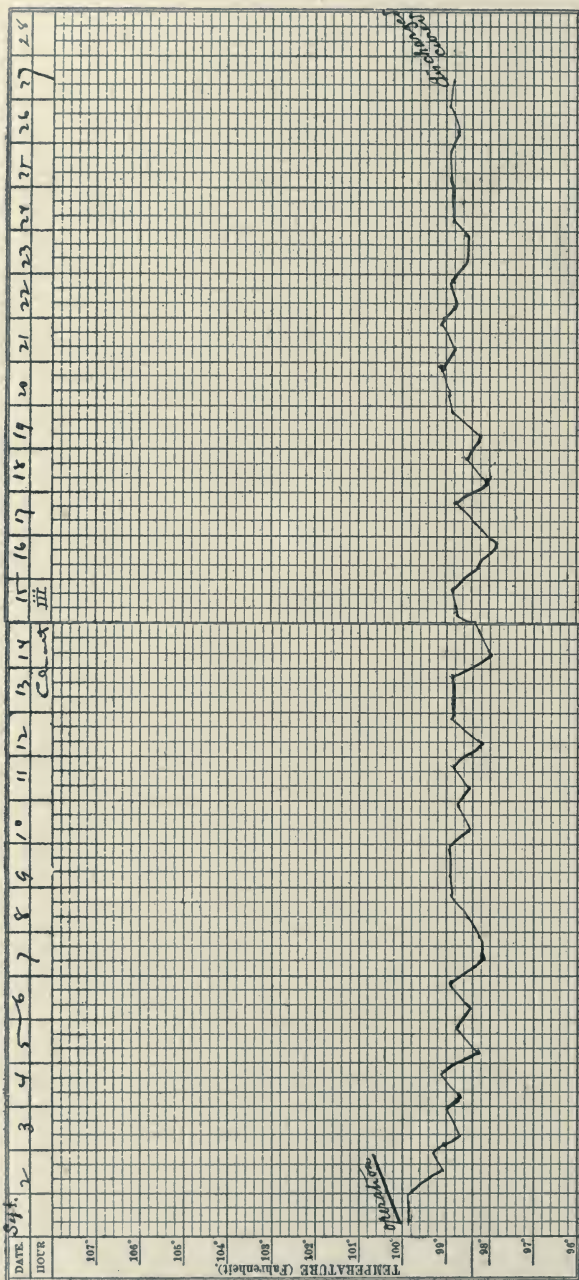
Professor of Diseases in Children, Visiting Physician to the New York Post-Graduate and German Hospitals, etc., New York.

Assuming that in a given case of pyothorax the proper surgical procedure has been accomplished we might expect, where the temperature has been high before operation, a fall of temperature and a normal or nearly normal temperature curve during



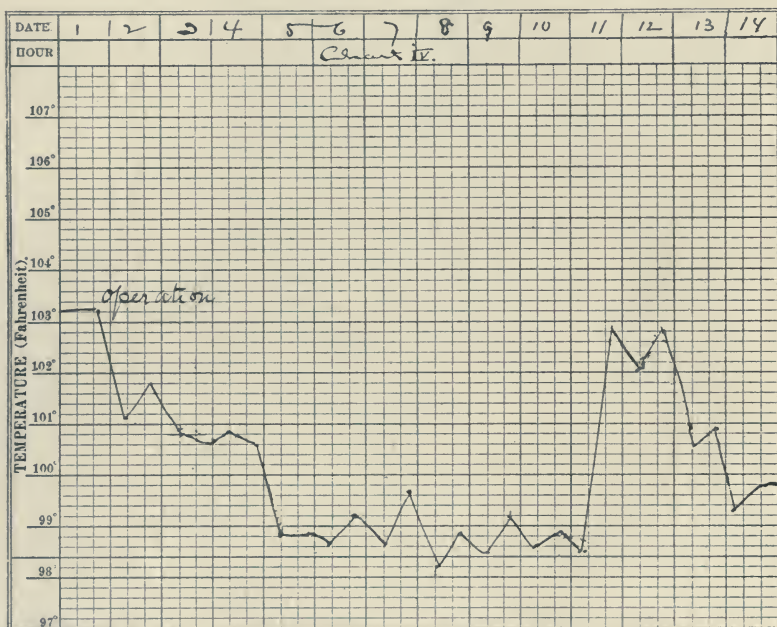
I. B. —. BOY FOUR YEARS OLD. ACUTE PYOTHORAX OF RIGHT SIDE. RESECTION OF RIB AND DRAINAGE. NORMAL TEMPERATURE ON THE DAY FOLLOWING THE OPERATION. REMOVAL OF DRAINAGE TUBE ON THE FIFTH DAY AND COMPLETE CLOSING OF THE WOUND ON THE NINTH DAY. NO FEVER TEMPERATURE AT ANY TIME AFTER OPERATION.

convalescence. It is well known, however, that on the contrary and in a large number of cases, a rise from the normal takes place at once or at various times during the further course of the disease and as this fever temperature almost invariably indicates



III. I. M. MALE. NO RISE OF TEMPERATURE ABOVE NORMAL IN TWENTY-SEVEN DAYS.

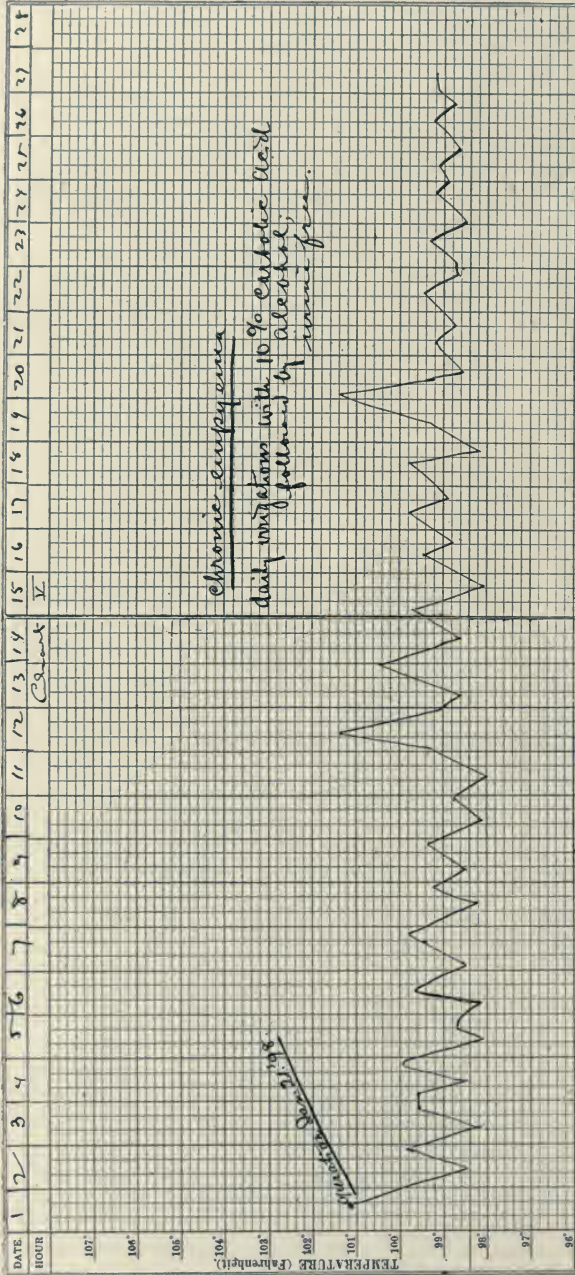
an extension of the disease or a complication of some kind, grave or trivial, it becomes a matter of great importance to correctly interpret such fever and remove any source of irritation if present, and if possible, to remedy, before the patient is exhausted. The correct interpretation of fever after empyema operation is, in some instances, extremely simple, and in others extremely difficult—so that it is nothing unusual for surgeons



IV. J. B. BOY OF FIVE YEARS. IN THIS CASE THE TEMPERATURE BECAME NORMAL ON THE FOURTH DAY AFTER RESECTION. ON THE ELEVENTH DAY AFTER OPERATION A RISE TO 103° F. WAS NOTICED, AND ON EXAMINATION IT WAS FOUND THAT THE DRAINAGE TUBE HAD SLIPPED INTO THE THORACIC CAVITY. AFTER RECOVERY OF THE TUBE THE TEMPERATURE FELL AND REMAINED NORMAL.

with but little clinical experience to inquire simply into the matter of drainage and, if such appears satisfactory, to overlook other factors equally important.

With a view of eliciting discussion on this subject before the American Pediatric Society, the writer has carefully followed the histories of eighty-six private cases and also the cases treated in the Babies' Ward, Post-Graduate Hospital, and others operated upon in the children's clinics of the school, in all about three



V. L. B. A GIRL OF SEVEN YEARS. THIS PATIENT HAD HAD THREE OPERATIONS FOR PYOTHORAX BEFORE ENTERING THE POST GRADUATE HOSPITAL. A FOURTH OPERATION WAS PERFORMED AND THE ABSCESS CAVITY WAS IRRIGATED WITH 2 PER CENT. CARBOLIC ACID WATER AND SUBSEQUENTLY WITH 10 PER CENT. CARBOLIC ACID SOLUTION AND ALCOHOL. NO INTOXICATION SYMPTOMS WERE NOTICED AND A STEADY IMPROVEMENT TOOK PLACE.

hundred cases. An endeavor will be made to show in a brief manner in what extent a careful study of body temperature will aid us in establishing the causative factor in fever temperatures, following operations for pyothorax.

It may be admitted at the outset, that a completely afebrile course is exceptional. Charts I., II., and III. will illustrate that small group of cases in which the temperature drops to normal after operation, and remains about normal. Such cases are not of long standing, and the pus is localized by adhesions and the healing process is a rapid one.

In chart IV., the otherwise normal temperature curves shows an elevation eleven days after operation, due to irritation of a lost drainage tube, which had accidentally slipped into the pus cavity of the thorax. If drainage from the pus cavity is interfered with in consequence of granulations or from other causes, a rise of temperature will almost invariably result.

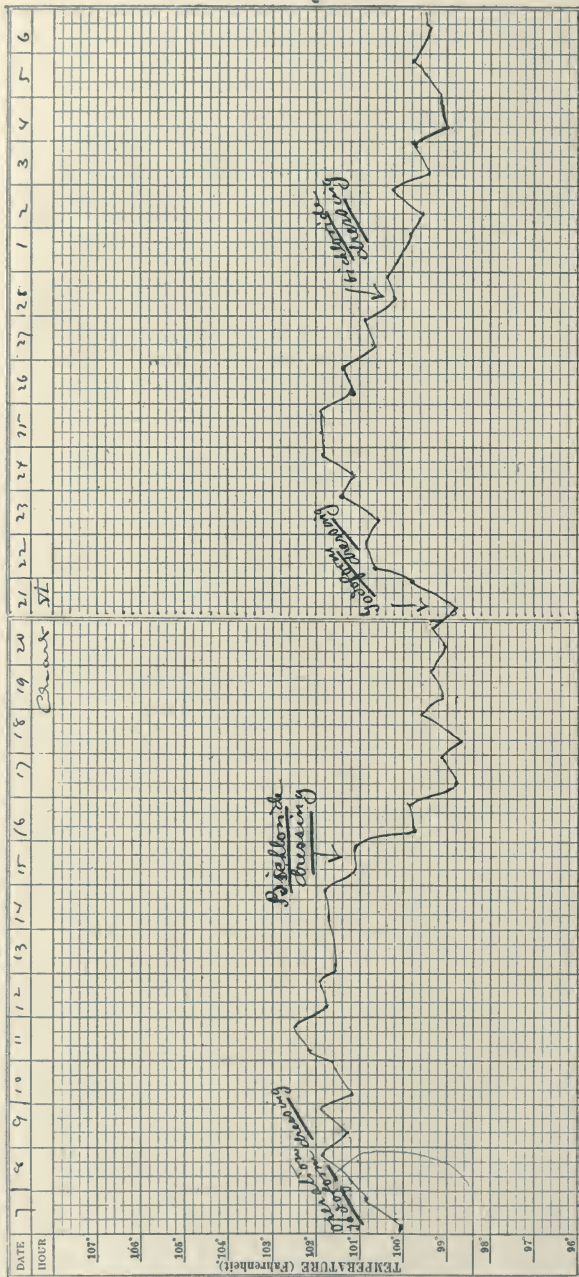
Instances of a rise of temperature from intoxication with *iodoform* or *carbolic acid* are not rare, and are sufficiently characteristic to be readily detected. Carbolic acid produces the well-known smoky urine with some fever. Irrigation of the abscess cavity is practiced but very little, and carbolic acid is not employed much for this purpose except in special cases.

As an evidence of the tolerance of carbolic acid, owing to the non-absorbing condition of a thick membrane, I present chart V., which shows an almost normal temperature, free urine and steady improvement in a case of chronic empyema of long standing in a girl of seven, whose thoracic abscess was irrigated with 10 per cent. carbolic acid solution and pure alcohol daily for a long period.

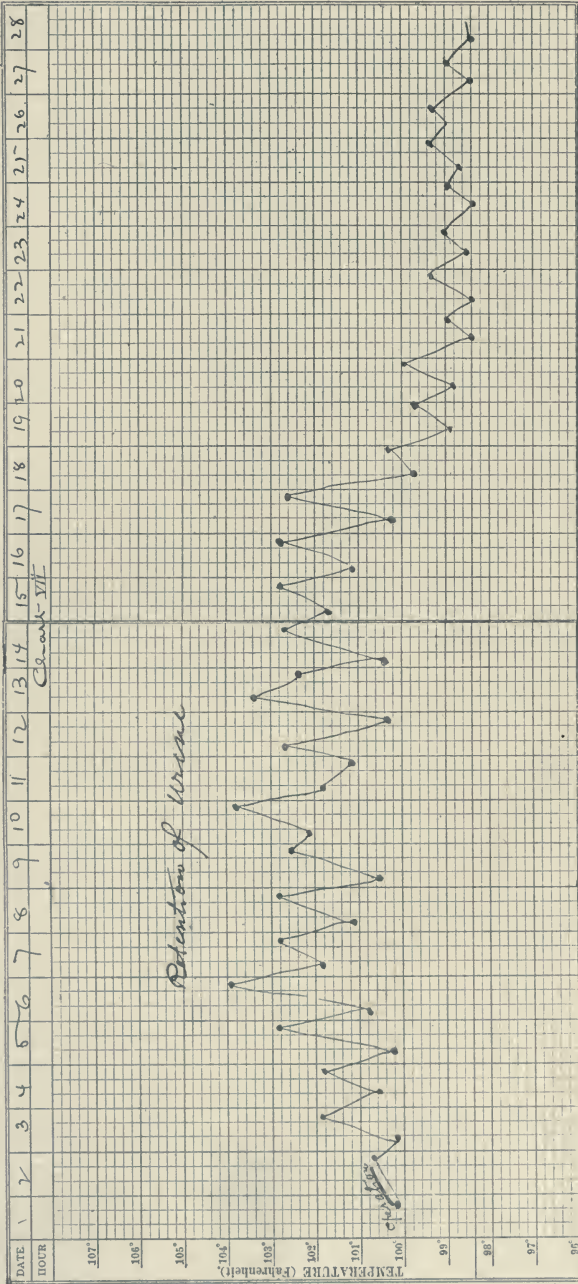
To illustrate the toxic effects of *iodoform*, I present chart VI. This complication may be recognized by fever, nausea or vomiting and frequently a scarlatiniform rash, all symptoms disappearing when the drug is no longer used.

The cases showing a rash are very much like true scarlatina, and it is not always possible to distinguish one from the other, for we find a punctate and acuminate rash in true scarlatina as well as in intoxication rashes.

Chart VII. illustrates a case in which retention of urine was the obvious cause of a fever temperature. The rise in temperature took place right after the operation, and a retention of urine necessitated the use of the catheter twice daily for eleven days.



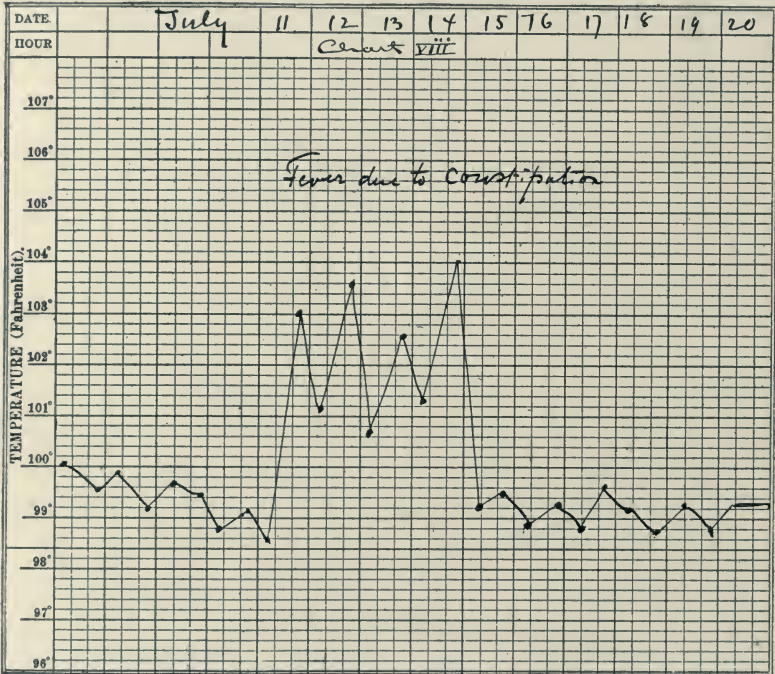
VI. A. G. FOUR YEARS. OPERATION FEBRUARY 7TH, FOLLOWED BY RISE OF TEMPERATURE, NAUSEA, VOMITING. IODOFORM DRESSING DISCONTINUED AND BI-CHLORIDE DRESSING SUBSTITUTED, FEBRUARY 14TH. THE TEMPERATURE SOON BECAME NORMAL AND THE CHILD TOOK FOOD. FEBRUARY 21ST THE IODOFORM DRESSING WAS AGAIN APPLIED AND THE SAME PHENOMENA WERE OBSERVED. THE IODOFORM DRESSING WAS DISCONTINUED AGAIN ON FEBRUARY 26TH, AND THE CHILD AFTERWARDS DID WELL. BETWEEN THE 21ST TO 26TH OF FEBRUARY A PUNCTATE SCARLATINIFORM RASH WAS ALSO NOTICED. THERE WERE NO THROAT SYMPTOMS AND SUBSEQUENTLY NO DESQUAMATION.



VII. M. D. A GIRL OF THREE YEARS. THE TEMPERATURE ROSE AFTER THE OPERATION AND RETENTION OF URINE WAS NOTICED WHICH NECESSITATED THE USE OF THE CATHETER TWICE A DAY FOR ELEVEN DAYS. ON THE TWELFTH DAY THE TEMPERATURE DROPPED TO NORMAL AND THE CATHETER WAS NO LONGER NECESSARY. DURING THE TIME THERE WAS GOOD DRAINAGE FROM THE THORAX AND NO COMPLICATIONS. THE REMOVAL OF THE DRAINAGE TUBE HAD NO INFLUENCE UPON THE TEMPERATURE. THERE WAS EVIDENTLY SOME OBSCURE CONNECTION BETWEEN THE FEVER AND THE RETENTION OF URINE. THERE WAS NO MALARIA.

On the twelfth day, the urine was passed voluntarily and the temperature dropped to normal. During this time there was good drainage from the thorax and no complications could be detected. The bladder was not infected, the removal of the drainage tube had no influence upon the temperature; there was no malaria nor nephritis, but evidently some obscure connection between the fever and retention of urine.

Constipation as a cause of fever is well illustrated by chart



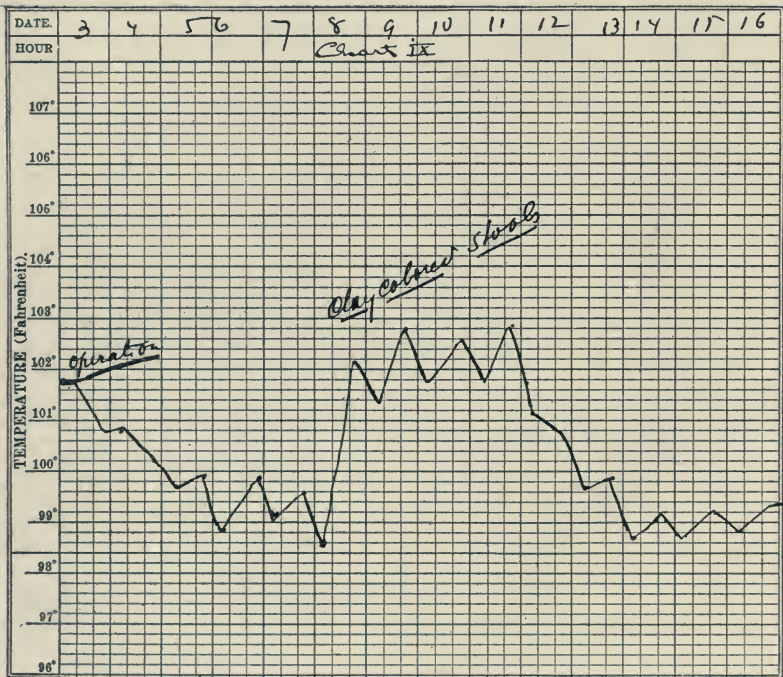
VIII. R. B. A GIRL OF FIFTEEN MONTHS. OPERATION JUNE 19, 1895. NORMAL TEMPERATURE UNTIL JULY 11TH. FOUR DAYS' FEVER DURING CONSTIPATION AND AFTER A BRISK LAXATIVE A FALL OF TEMPERATURE TO NORMAL, WITH NORMAL CONVALESCENCE.

VIII., in which case an operation for pyothorax was performed on June 9, 1895, with a normal temperature until July 11th, then high fever with constipation for four days and normal temperature and normal convalescence after a brisk purge.

In the case of James Curtis, four and one-half years old, a rise of temperature was noticed simultaneously with the appearance of clay stools and a jaundiced color (chart IX), all of which rapidly disappeared.

Secondary and extra thoracic abscesses as a cause of fever are frequently met with and I have quite a list of such cases, small and large abscesses developing in axilla, buttocks, groin, parotid gland, also extra pleural abscesses and otitis media.

To illustrate this group, I will cite the case of S. S., whose temperature range is shown by chart X. She underwent operation at the age of two years for pyothorax, and remained well for three years, when she developed pyothorax of the same side and



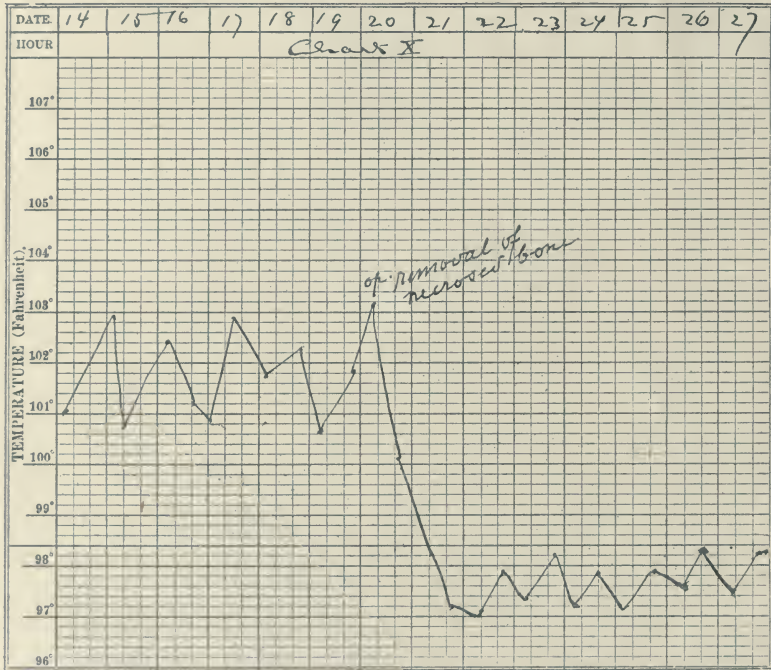
IX. J. C. INCISION FOR PYOTHORAX, JANUARY, 1897. RESECTION OF RIB, FEBRUARY 3D, AFTER WHICH THERE WAS NORMAL TEMPERATURE UNTIL FEBRUARY 8. MODERATE FEVER FOR THREE DAYS, WITH SYMPTOMS OF GASTRIC DISTURBANCE AND CLAY COLORED STOOL.

had irregular fever from September, 1897, to January 20, 1898, on which day a portion of necrosed rib was removed and the patient made a rapid afebrile recovery.

To this group also belong two cases of extra thoracic abscess in the axillary region in which, after incision, no fall of temperature took place. A more careful examination elicited the

presence of pyothorax of the same side which had been overlooked. Sub-diaphragmatic and lung abscesses consequent upon pyothorax are also met with, but present no characteristic fever curve.

We now come to consider a group of cases in which convalescence is disturbed by a distinct specific infection or contagion, such as measles, scarlatina, erysipelas, diphtheria, specific vulvo-



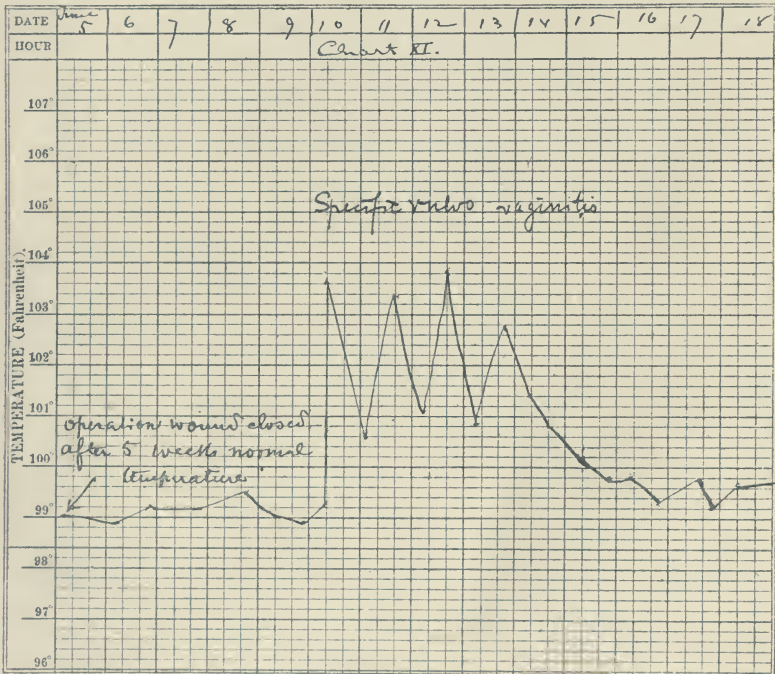
X. S. S. A GIRL OF FIVE YEARS. REMOVAL OF NECROSSED RIB SECONDARY TO EMPYEMA, FOLLOWED BY FALL OF TEMPERATURE AND NORMAL CONVALESCENCE. AT THE AGE OF TWO THIS PATIENT HAD PYOTHORAX OF THE SAME SIDE, FOR WHICH AN OPERATION WAS PERFORMED AT THE POST GRADUATE HOSPITAL, AND WHICH HEALED COMPLETELY. THREE YEARS AFTER THIS ATTACK, A SECOND ATTACK ON THE SAME SIDE TOOK PLACE, AS NOTED ABOVE.

vaginitis, and the following charts will illustrate some of the conditions here cited:

Chart XI. J. F., four years old. After five weeks of nearly normal temperature, the operation wound closed June 5th. On June 10th a sudden fever temperature, lasting four days, was noticed, and a most careful examination at first failed to locate the source of fever. Finally an offensive purulent vulvo-vaginal dis-

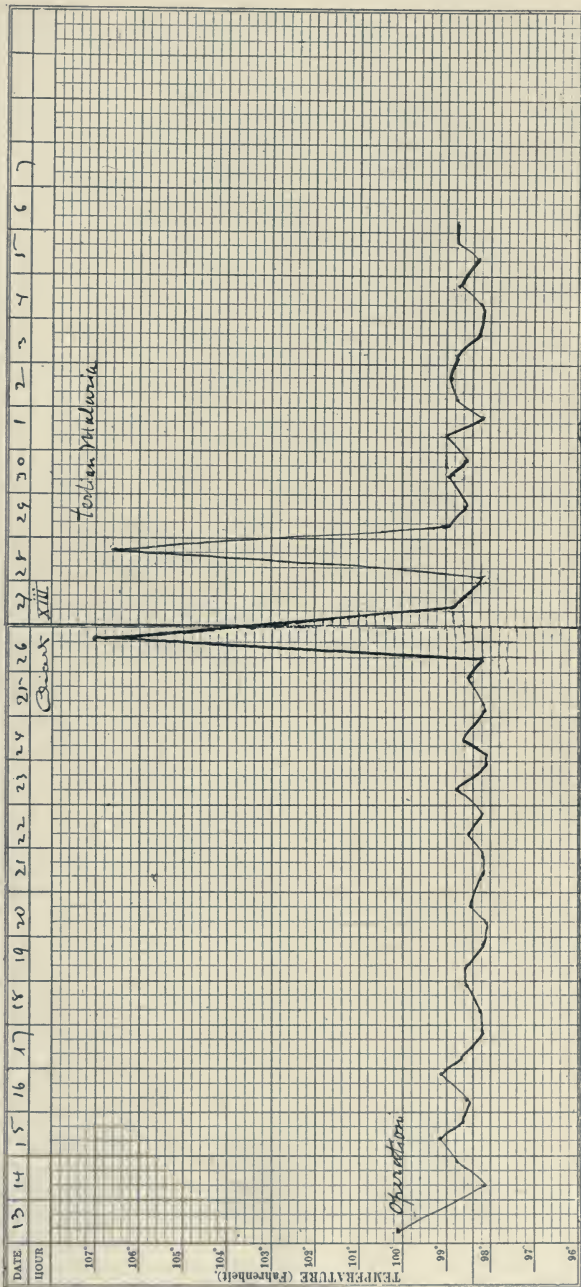
charge was detected containing gonococci. The parts were inflamed and bleeding to the touch, and energetic local treatment was followed by local improvement and defervescence.

Chart XII. shows a diphtheritic infection in a case of pyothorax—G. B., eighteen months. Operation April 19. On May 8th a sudden rise of temperature to 105° F. was noticed, due to diphtheritic sore throat, which subsided promptly after administering 1,000 units of antitoxin, the temperature thereafter remaining normal.



XI. J. F. A GIRL OF FOUR YEARS. AFTER FIVE WEEKS' NORMAL TEMPERATURE THE OPERATION WOUND CLOSED JUNE 5TH. ON JUNE 10TH A SUDDEN FEVER TEMPERATURE LASTING FOUR DAYS WAS NOTICED. ON CAREFUL EXAMINATION IT WAS FOUND THAT A SPECIFIC VULVO-VAGINITIS WITH INTENSE INFLAMMATION OF THE PARTS WAS RESPONSIBLE FOR THE FEVER, WHICH SUBSIDED AFTER APPROPRIATE LOCAL TREATMENT WAS INSTITUTED.

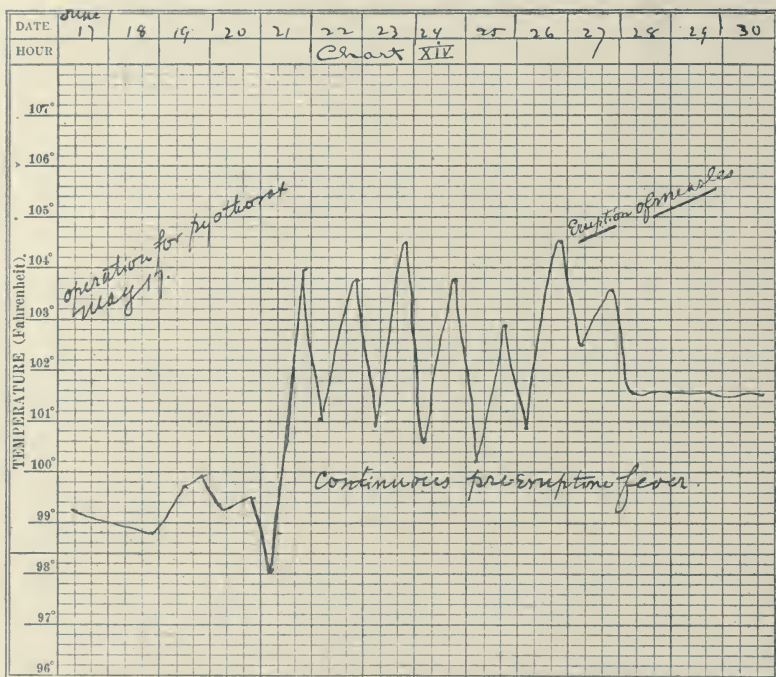
Chart XIII. shows a typical malarial febrile attack. The patient, a boy three years old, was operated upon for pyothorax April 13, 1897, and ran a very normal temperature to the 26th of April, when 107° F. was registered, followed by a drop to 98° and a rise to 106° on the 28th. A tertian malaria which was



XIII. J. M. A BOY OF THREE AND A HALF YEARS. MALARIAL INFECTION DURING THE CONVALESCENT STAGE OF PYOTHORAX. PLASMODIUM FOUND.

necessary to reproduce them here. However, it may be stated that the recognition of complications presented in this group is made without any difficulty, because the characteristic rash or throat exudate will make its appearance within two to six days from the time of the temperature rise. In the absence of a culture test, we are not able to distinguish positively a mild case of scarlatina from a scarlatiniform rash.

We will now consider that class of cases in which there is



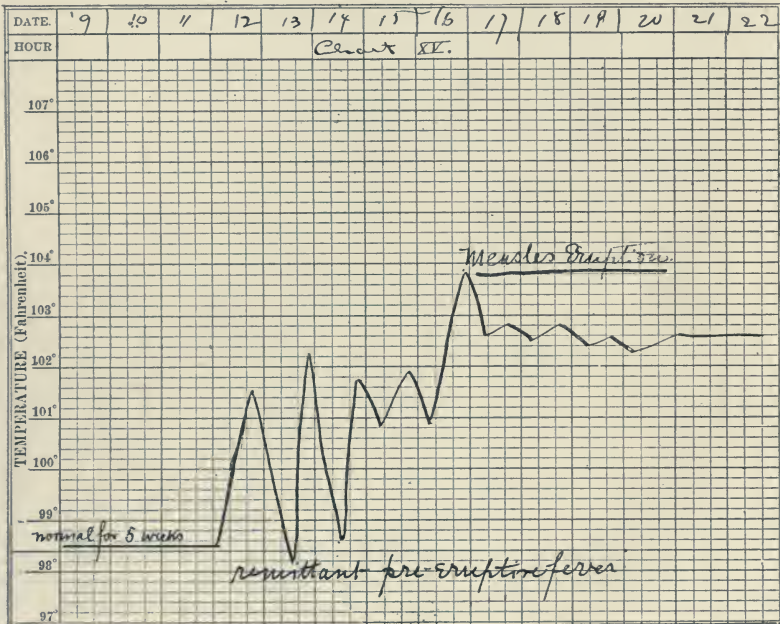
XIV. W. K. MALE. OPERATION MAY 17, 1895. NORMAL TEMPERATURE TO JUNE 21ST, ON WHICH DAY THERE WAS A RISE TO 104° F., AND SLIGHT COUGH. JUNE 26TH, ERUPTION OF MEASLES.

but a slight remission of fever after operation and where we may have an extension of the inflammatory process to the other side of the chest, or to the pericardium, or peritoneum, or concomitant lobar, broncho or tuberculous pneumonia.

Chart XVI. illustrates the bilateral invasion of the chest in a boy three years old (G. L.), who was operated upon for left-sided pyothorax, September 13th, the temperature remaining low until October 3d, on which day a rise to 105° was registered

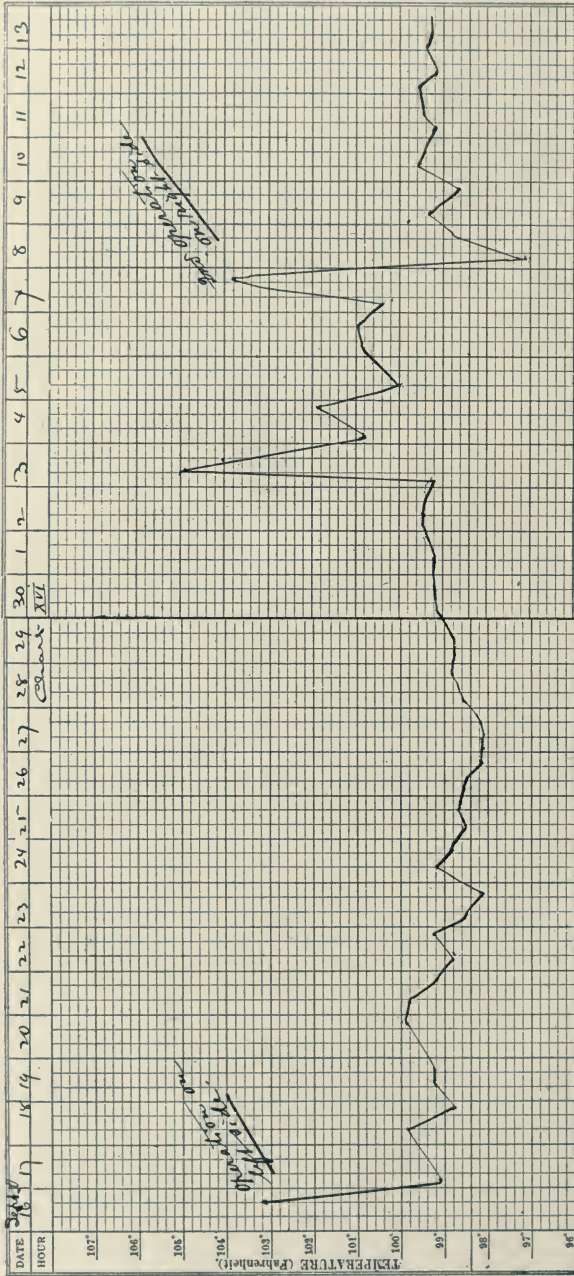
with renewed rapid respiration. The aspirating needle showed sero-pus on the right side and a second operation was followed by a drop to the normal.

A similar case is that of R. D., eighteen months old, *Chart XVII.* In this case a rapid invasion of both sides took place first right and then left, with but a few days intermission between the attacks, as shown by the temperature curve. In all such cases



XV. M. R. A GIRL OF FIVE YEARS. NORMAL TEMPERATURE FROM NOVEMBER 6TH TO DECEMBER 12, 1897. ON DECEMBER 12TH, RISE OF TEMPERATURE FROM $97\frac{1}{2}^{\circ}$ TO $101\frac{1}{2}^{\circ}$ AND DROP TO NORMAL ON THE FOLLOWING DAY, AND REPETITION OF THE SAME PHENOMENON ON THE NEXT DAY, AFTER WHICH THERE WAS CONTINUOUS FEVER TILL DECEMBER 16TH, ON WHICH DAY THE ERUPTION OF MEASLES APPEARED, THUS SHOWING THAT IN THE INCUBATIVE OR PRE-ERUPTIVE STAGE OF MEASLES THE FEVER MAY DROP TO THE NORMAL AT DIFFERENT TIMES OR IT MAY SHOW THE CONTINUOUS TYPE, AS IN CHART XIV.

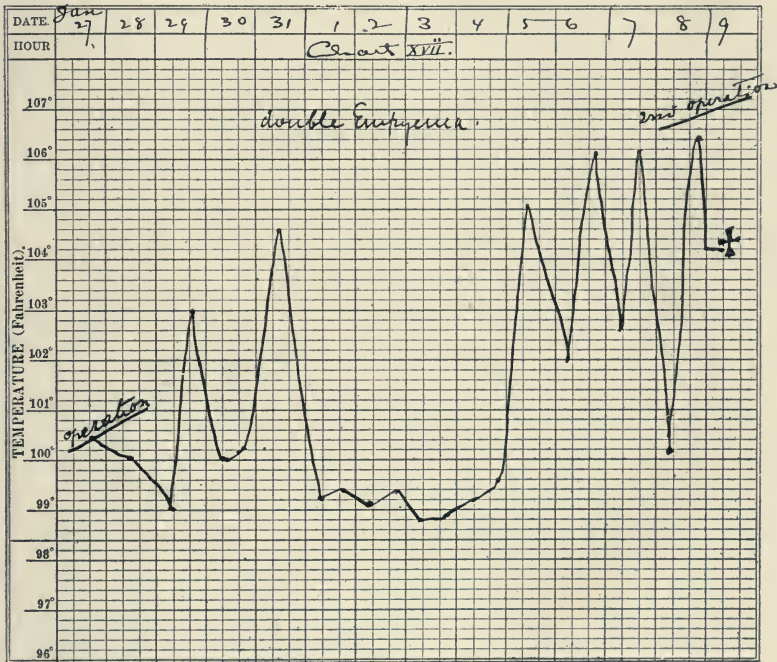
the fever and rapid respiration and physical signs of fluid will readily establish the diagnosis which is confirmed by the aspirating needle. To correctly interpret those cases in which the fever is kept up by a complicating unresolved lobar pneumonia or broncho-pneumonia or tubercular pneumonia is by no means easy, particularly if dense adhesions are present and give dulness on percussion. The unresolved lobar pneumonia will give dulness



XVI. Z. L. THREE YEARS. DOUBLE EMPYEMA. ONE OPERATION SEPTEMBER 16, 1894. NORMAL CONVALESCENCE AND LOW TEMPERATURE UNTIL OCTOBER 3D. ON THIS DAY A RISE OF TEMPERATURE TO 105° OCCURRED AND RENEWED EMBARRASSMENT OF RESPIRATION, WITH GOOD DRAINAGE ON THE OPERATED SIDE. THE RIGHT THORAX WAS FOUND FULL OF SERO-PUS, AND AFTER ITS REMOVAL THE TEMPERATURE DROPPED TO SUBNORMAL.

and bronchial breathing and the temperature will not be as high as in the other varieties just cited.

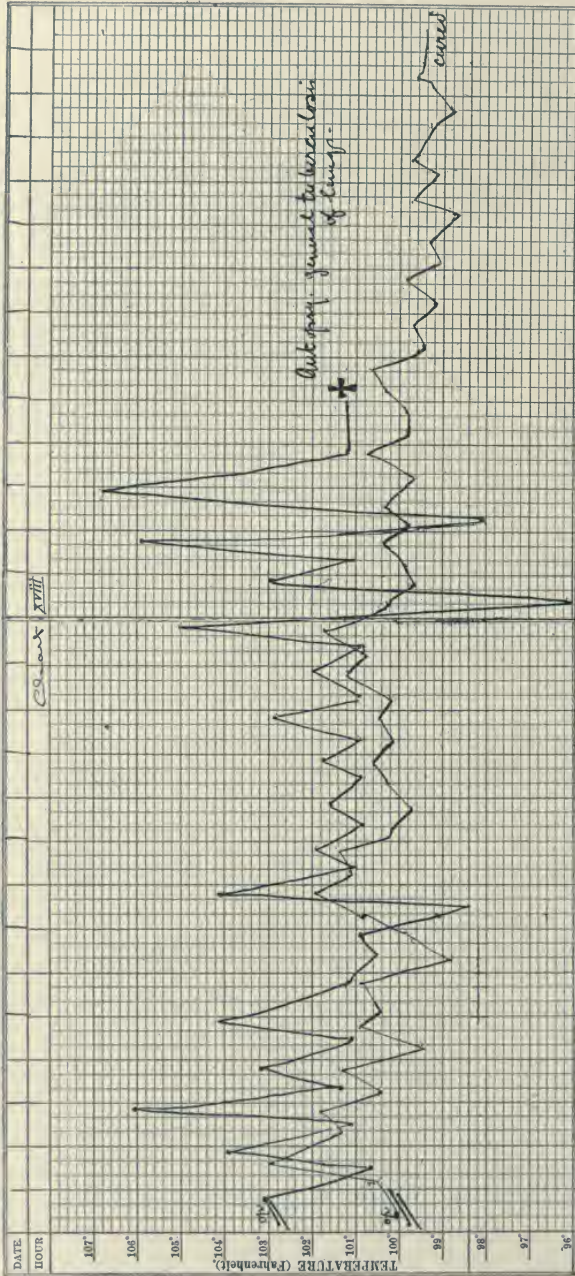
To distinguish a broncho-pneumonia from a general pulmonary tuberculosis by physical signs and symptoms in the absence of bacillary proof, is rarely possible. A pulmonary miliary tuberculosis may be suspected from the high septic temperature, rapid breathing, diarrhœa, loss of weight. A careful and re-



XVII. R. D. A BOY OF EIGHTEEN MONTHS. OPERATION ON RIGHT SIDE, JANUARY 27TH. FEVER CONTINUED BUT RESPIRATION DROPPED FROM 72 TO 32 FROM DAY OF OPERATION TO FEBRUARY 6TH, ON WHICH DAY THE RESPIRATION ROSE TO 76 AND THE TEMPERATURE BECAME VERY HIGH. THERE WAS GOOD DRAINAGE ON PRIMARY SIDE BUT THE LEFT SIDE NOW FILLED WITH SERO-PUS, WHICH WAS ÉVACUATED. THE PATIENT DIED ON FEBRUARY 9TH.

peated examination of the pus in tuberculous pyothorax will occasionally reveal the presence of tubercle bacilli.

In the following, *Chart XVIII.*, the temperature curves of tuberculous pyothorax and empyema with unresolved pneumonia are shown together for comparison. *It will be noticed that the excursions of the mercury are much greater in tuberculous pyothorax than in that form complicated with unresolved pneumonia, and the average fever curve is higher, and the writer has*



XVIII. DOUBLE TEMPERATURE CHART SHOWING PYOTHORAX WITH UNRESOLVED PNEUMONIA OF SAME SIDE AND PYOTHORAX WITH PULMONARY TUBERCULOSIS.

found this relative difference in the temperature curve almost constant in similar cases. Chart XIX. is another illustration of the peculiar temperature curve in tuberculous pyothorax.

The temperature curve in pyothorax with putrid bronchitis or gangrene of lung is not different from that of any septic temperature curve, although the writer has seen three such cases in which the temperature was almost normal under free drainage, the discharging pus and sputum being brownish in color and extremely offensive to the smell.

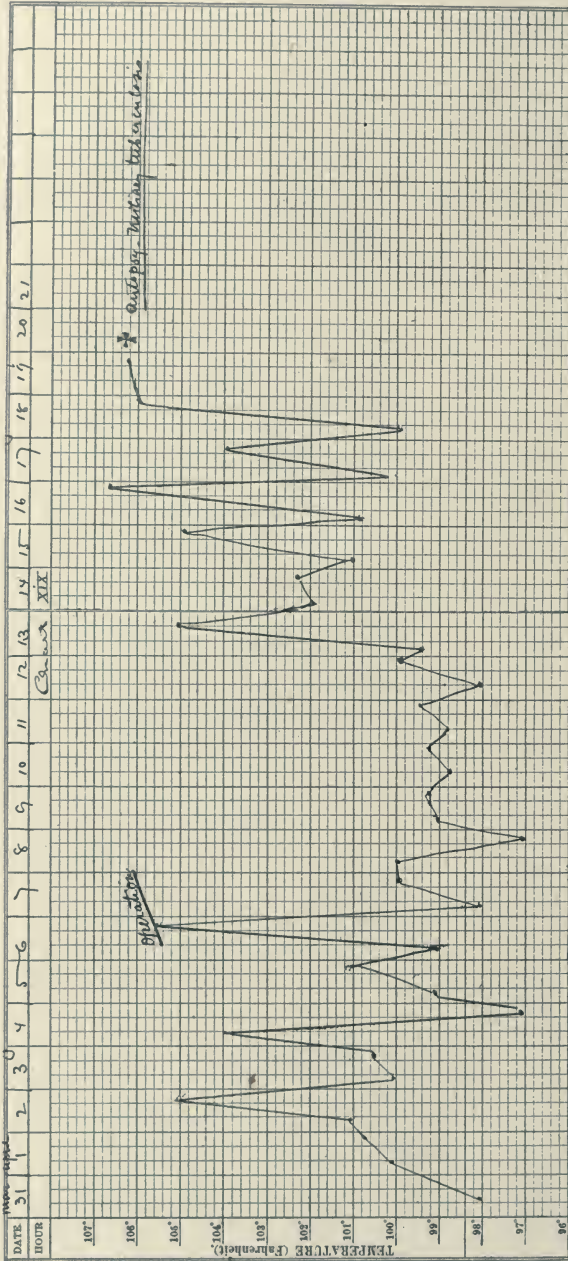
In cases of tuberculosis of the lung with pulmonary abscess of long standing, with subsequent formation of an empyema, there is usually no satisfactory drop of temperature after operation, and in *chronic empyema with a localized abscess cavity and insufficient expansion of the lung in consequence of adhesions an afebrile course may persist for a long time if drainage is sufficient, but finally the pus microbe will infect other organs, the kidney and intestines, such complication presenting sufficient symptoms in addition to the rise of temperature to make them easy of recognition. Cases of this nature terminating fatally often show amyloid degeneration on autopsy.*

Chart XX. illustrates a case of pyothorax which did well for four weeks and suddenly developed a high temperature and a bloody diarrhœa, with 20 per cent. albumin by bulk in the urine.

There is another variety of pyothorax cases which may be termed the acute septic cases, as frequently met with in very young children. Charts XXI. and XXII. will illustrate such conditions: L. S., two years old, was operated upon November 27th, and on November 29th pus was found on the other side of the thorax and also removed by incision. The temperature was 104° at the operation and remained high till death. The autopsy showed besides bilateral pyothorax, broncho-pneumonia in both lungs and acute nephritis.

The next, Chart XXII., shows the acute septic course of pyothorax in a very young child. The writer has observed eight cases below the age of six months. Such patients rarely recover, even with good drainage and good lung expansion. A high septic temperature usually persists and pulmonary œdema ends the scene.

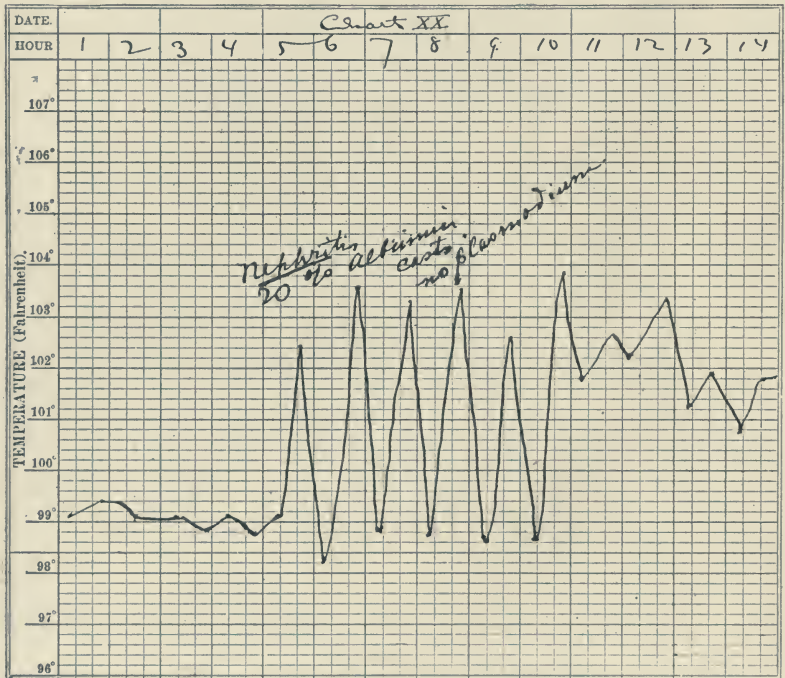
To this category of septic cases, also belongs the following: H. S., fourteen months; operation November 26th. A normal temperature, irregular slight fever until December 21st, on which



XIX. M. F. A GIRL OF SEVEN WEEKS. VERY HIGH TEMPERATURE BEFORE OPERATION. AFTER OPERATION FOUR DAYS OF AFEBRILE TEMPERATURE, THEN HIGH FEVER AGAIN UNTIL DEATH. THE PULMONARY SYMPTOMS WERE THOSE OF BRONCHO-PNEUMONIA. THE TEMPERATURE CURVE SPEAKS FOR TUBERCULOSIS, WHICH WAS FOUND AT AUTOPSY.

day the temperature rose to 105°. The color of the pus changed to green and the culture test showed the presence of the bacillus pyo-cyaneus. There was no pulmonary complications and no diarrhœa, but some vomiting, and death resulted. No autopsy. The infection by the green pus microbe must be looked upon as the cause of the high septic temperature.

Tubercular and cocci meningitis is occasionally met with as a complication of pyothorax. The accompanying temperature is

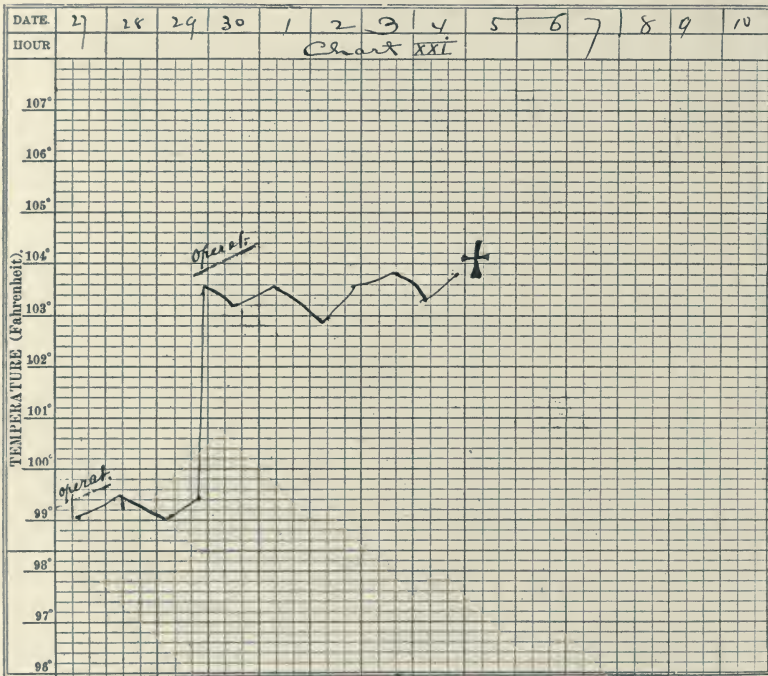


XX. L. T. A BOY OF FOUR YEARS. OPERATION APRIL 14, 1898. TEMPERATURE NORMAL TILL MAY 5TH, WHEN FEVER, WITH DAILY REMISSION TO NORMAL, APPEARED, WITH 20 PER CENT. BY BULK ALBUMIN IN URINE, AND BLOODY CASTS AND BLOODY DIARRHŒAL STOOLS.

not sufficiently characteristic to establish diagnosis, which, however, may be arrived at by a study of the objective manifestations.

We now come to the most important class of cases from the practical standpoint in which, after operation, thorough drainage and no appreciable complications, the irregular septic temperature persists, is due to *deep seated multilocular accumulation of pus* or abscesses not reached by the primary operation.

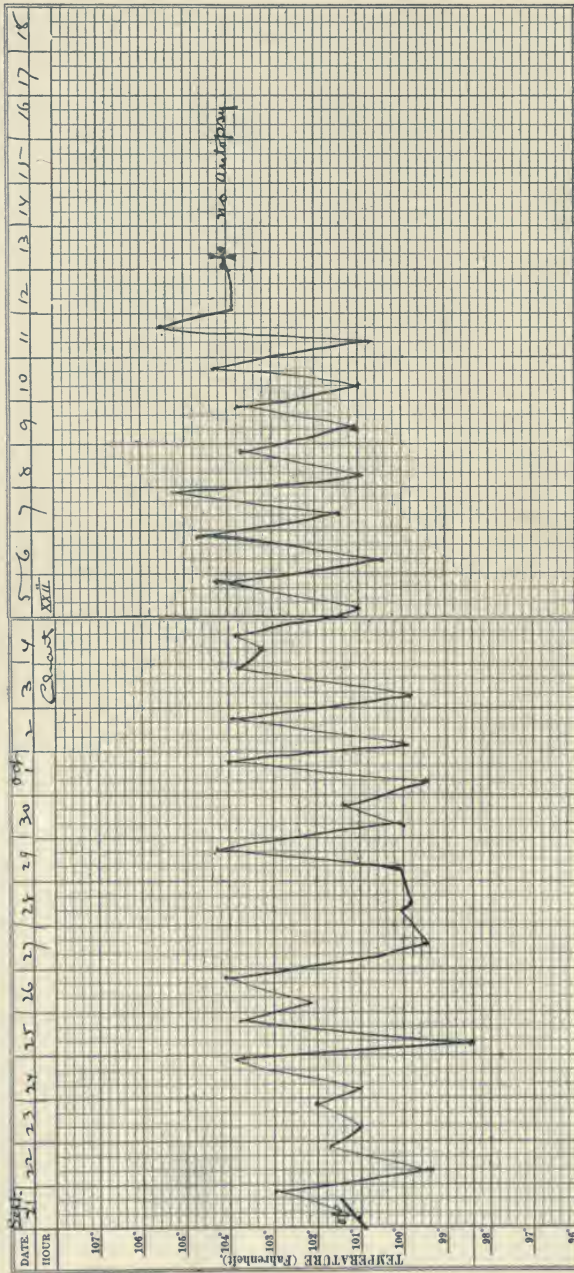
Of such cases not a few have come under the writer's notice, some in which the condition was recognized at the time of operation and a few in which after operation, notwithstanding good drainage and no complications and fair general condition, the temperature would rise to 103° or 104° at irregular intervals. In one instance even the aspirating needle in the hands of three very able physicians and surgeons and introduced at various times in the course of three weeks, failed to detect a deep seated pus de-



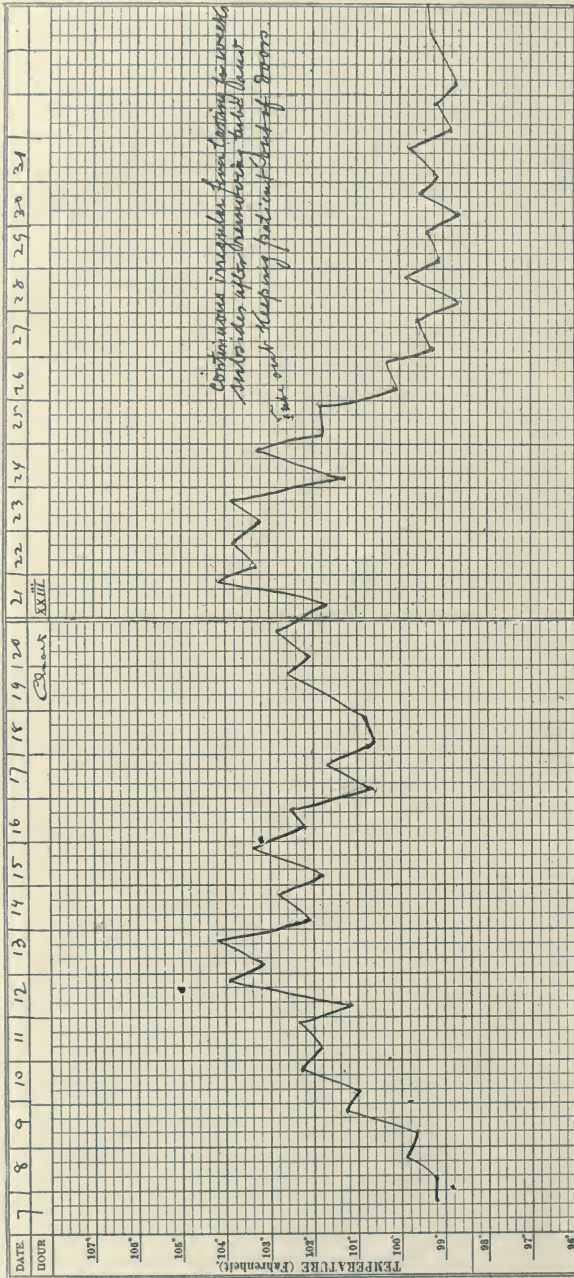
XXI. L. S. A BOY OF TWO YEARS. DOUBLE PYOTHORAX; ACUTE SEPSIS; DEATH AND AUTOPSY FOUR DAYS AFTER SECOND OPERATION; BRONCHO-PNEUMONIA FOUND IN BOTH LUNGS; ACUTE NEPHRITIS. NO FALL OF TEMPERATURE AFTER OPERATION.

posit, which was strongly suspected and which finally found its way to the chest wall, where it was eventually recognized and drained four weeks after the first operation, after which the temperature fell and remained normal.

Finally I will draw attention to certain cases which do quite well for two or three weeks after operation and then show an irregular rise of temperature. The discharge from the tube has almost ceased. No complications can be detected by examin-



XXII. I. C. A BOY OF EIGHT MONTHS. SEPTIC TEMPERATURE AFTER OPERATION; GOOD DRAINAGE; LITTLE COUGH; NO SLEEP; VOMITING; BLOODY STOOLS.



XXIII. W. F. A GIRL OF FIVE YEARS. OPERATION APRIL 3, 1897. ALMOST NORMAL TEMPERATURE TO MAY 9TH, THEN DAILY FEVER RANG-
 ING FROM 101° TO 104°. MAY 25TH THE TUBE WAS REMOVED AND THE CHILD WAS CARRIED OUT OF DOORS; WITHIN ONE WEEK THE EXTREME
 ANEMIA IMPROVED AND THE TEMPERATURE FELL AND REMAINED NORMAL.

ing the urine, the blood and the various organs. If in this class of cases we remove the *drainage tube* and have the patient taken out doors in spite of the fever, we get a rapid and satisfactory improvement. These cases remind us of the scarlatina patients with fever lasting for weeks after the rash has disappeared, and for which the closest examination will fail to locate a cause. The probability is, that such temperatures are occasioned by what we understand to be the result of absorption of inflammatory products. The drainage tube is in itself a source of irritation and should be removed as early as possible. Should a reaccumulation of pus take place and the wound be closed, it is a simple matter to enter the original cavity through the soft parts, if resection of a rib segment has been done. *Chart XXIII.* illustrates such a case. Fever sets in five weeks after operation, continues for three weeks, and subsides as soon as drainage tube is removed and child is carried out of doors.

In conclusion I would remark that our investigation has shown that in the vast majority of cases a rise of temperature is observed at some time or other after operations for pyothorax, and that only in a very few cases the temperature remains normal or thereabout until healing is accomplished.

This rise of temperature may mean very little, *but it always indicates something which we should endeavor to locate and correct.* The successful management of pyothorax is not alone a matter of incision and drainage, but calls for accurate clinical investigation and observation. A carefully kept record of temperature furnishes valuable evidence as the underlying cause of fever.

753 MADISON AVENUE.

IRRIGATION BY SUBMERSION IN THE TREATMENT OF EMPYEMA.

BY SAMUEL S. ADAMS, A.M., M.D.,

Professor of Pediatrics, University of Georgetown; Visiting Physician
Children's Hospital, Washington, D. C.

To A. Zeman must be given the credit of introducing the warm bath as a means of washing the pleural cavity in cases of pyothorax.

In the *Voyenno Med. J.*, (*Army Med. J.*), 1896, clxxxvii., 822-829, November, Zeman gives the most interesting account of the accidental discovery of this method, in an article entitled "Washing the Cavity of the Pleura in Empyema by means of Baths."

He considered the usual method of washing out the pleural cavity unsatisfactory, as only the liquid matter is removed, while many products of inflammation adhere to the pleura, thus keeping up the irritation. The author accidentally hit upon the following method of washing out the pleural cavity:

CASE I. G. L. entered the Military Hospital March 15, 1893, suffering from empyema of the right side. The author performed thoracotomy, with resection of a rib. A great quantity of pus escaped and the cavity was irrigated with a physiological salt solution. On the following day the patient literally swam in pus. Having again washed the cavity with physiological salt solution three times, a bath was ordered to wash off the pus from the outside of the body. To prevent the pus from re-entering the pleural cavity the bath-tub was to be filled so as to reach below the opening when the patient was sitting in it. He was instructed to sit so as to prevent the water from entering the opening. He, however, desiring a thorough cleansing, allowed the water to go above the opening and into the pleura cavity, having taken advantage of a lack of vigilance of his attendants. At this moment Zeman appeared upon the scene and noticed that with each inspiration, in consequence of the expansion of the thorax and negative pressure within the pleural cavity, the water entered with great force, and was forcibly expelled by each expiration, carrying with it a great mass of thick pus and fibrinous coagula which had remained in the pleural cavity in spite of the

irrigation. He then placed his hand over the opening and it was drawn in by a force estimated to be equal to one-fifth of an atmosphere. From that time the patient was given daily baths. The ordinary river water was filtered and boiled, but no disinfectants were used. The water in the tub stood about three inches above the opening, and the temperature was 95° to 99.5° Fahr. The bath usually lasted from ten to fifteen minutes.

In two more cases the author verified the above. After operation he thoroughly washed out the pleural cavity with the irrigator until the water was returned clear; then he placed his patient in baths, and in both cases a mass of compact products of inflammation was washed out; but the difference was less marked after the first bath, because there were no longer masses of débris in the pleural cavity.

The fact that the force of the inflow of water from the baths equals one-fifth of an atmosphere, while much less force is employed with the irrigator, would explain the removal of the tough masses. In washing out with the irrigator the water does not reach every part of the pleural cavity; while, in accordance with Mariott's law, the water of the bath will reach every portion of the pleural surface. In the mechanism of this method there is sucking in and gushing out caused by inspiration and expiration, which in turn causes alternating positive and negative pressure, and hence a more thorough washing.

His reasons for preferring this method are:

1. It is simpler, cleaner, easier, and in the ten to fifteen minutes that the patient is sitting in the bath he will be washed out two to three hundred times; not three to four times.
2. Much economy is caused in dressings, as by removing the thickened matter from the pleural surfaces there will be less irritation and the production of much less pus.
3. The baths improve the general condition of the patient, causing better metabolism. In irrigation fresh water is constantly used; but as the pus and other products are heavier than water they fall to the bottom of the tub, while the upper portion of the water remains unpolluted.

CASE II. V. A., Sharpshooter, admitted January 9, 1894, with left-sided empyema. Thoracotomy: and resection of 8 cm. of seventh rib. Complete recovery in five weeks.

CASE III. A. V., male, aged two years, tuberculous, with left-sided empyema. Although the child was greatly exhausted,

3ctm. of a rib were resected and the child rapidly improved. On the ninth day, however, tubercular meningitis set in and the child died in a few days. Zeman says the last case should not be considered, as sufficient time was lacking in which to make a fair test of the treatment.

At least sixty baths were given in two of these cases and no bad effects were noted at any time.

This method of treatment was employed in the following case:

Mary M., aged eight years, Italian, was admitted to my service at the Children's Hospital, November 4, 1897. The following meagre history is all that could be learned from her mother and a woman that accompanied her, owing to their inability to understand and speak English. Her father and mother are in good health; and the family history does not show any transmissible taint.

Five months ago she is said to have had pneumonia of the right lung and has never recovered her health since the attack. The appetite is fair; she is constipated; cough has been almost incessant; and there has been occasional fever and night sweats. An abscess on the anterior aspect of the chest at the third interspace, to the right of the sternum, has appeared within the past five days.

She was placed on the table at my clinic and the following notes were taken. Emaciation marked; features pinched; slight icteric hue of skin; and cutaneous veins distended. Pulse weak and thready. An abscess about four inches in diameter is situated just to the right of the sternum between the second and fourth ribs. Its covering is very thin and its walls relax with each inspiration and become tense with expiration.

Respirations are accelerated but there is no dyspnoea. Flatness over entire chest anteriorly and posteriorly. Vesicular breathing absent. Bronchial breathing and voice anteriorly and posteriorly, but feeble and distant. Vocal fremitus almost absent. Bulging of intercostal spaces on right side.

The diagnosis was clear.

I made a free incision into the abscess, when more than a pint of greenish pus escaped and continued to do so with each expiration. A dressing was applied and she was transferred to the Surgical Service for operation.

November 8th. Dr. J. Ford Thompson resected about two

inches of the seventh rib in the right axillary line. A large quantity of pus escaped. The pleural cavity was thoroughly irrigated through the upper and lower openings, a drainage-tube having been inserted into the latter.

Her condition improved slowly and the physical signs of the normal chest were returning, but the profuse discharge continued in spite of thorough daily irrigation.

November 20th. It was determined to place her in a bath of boiled water at 100° Fahr. for fifteen minutes, after the method of Zeman. The cleansing was complete. With every inspiration the water would run into the two openings and, with expiration, would return laden with pus, which would sink to the bottom. Her entire body was kept under the water until the expiration gave out clear fluid, the time varying from ten to twenty minutes. At first a daily bath was given and then one every other day until she recovered and was discharged—the former January 1, and the latter February 21, 1898. She preferred the baths to irrigation with the syringe; sixteen baths were given, extending over three weeks. At the time of her discharge she was a rosy, robust girl, with a normal chest and normal chest sounds.

Having witnessed the thoroughness of this method of irrigation, and the comfort of the child while reposing in the warm bath, I must recommend it in cases of empyema in which an opening of sufficient size to enable free ingress and egress of the water is made. It is a matter of choice which antiseptic, if any, is to be used, but care should be exercised in employing those which are easily absorbed. Boiled water or a saturated solution of boric acid will be sufficient in most cases; the latter, however, would add greatly to the expense. The bath is prepared in the same manner as it is in the Brand method, and the water should be kept at a uniform temperature, 100° F., by adding warm water from time to time. There is no chilling, so the patient may remain in the bath several minutes after the water is returned perfectly clear.

This method also recommends itself in treating such cases in private practice, owing to the ease with which it can be carried out by the child's parents, as well as by its inexpensiveness.

DISCUSSION.

DR. WINTERS.—I think one feature of the method suggested is its simplicity and the fact that it does away with the resistance of the child to irrigation. There is nothing more unpleasant than the resistance of the child to irrigation after operation for empyema.

DR. CAILLÉ.—At the present time in the vast majority of cases, irrigation is not practised, but this seems to me to be a very pleasant way of cleansing the thorax in cases that require irrigation.

DR. ADAMS.—In tubercular disease this was tried but there was so much contraction of the chest that the water did not go in with any effect whatever.

DR. JACOBI.—There may be exceptional cases where the adhesions of the lungs are such that the entrance of very much water may interfere badly with the case.

ALBUMINURIA AS A LITHÆMIC MANIFESTATION IN EARLY LIFE.

BY B. K. RACHFORD, M.D.,

Clinician to Children's Clinic, Medical College of Ohio, Cincinnati.

The purpose of this paper is to note the importance and significance of the albuminuria which not infrequently is associated with certain lithemic paroxysms occurring in early life.

In other papers read before this society I have carefully described certain clinical pictures presented by lithemia, as it manifests itself in infants and children. In these papers I have called special attention to a group of symptoms from which these lithemic patients suffer, and have designated this symptom group as a "migrainous gastric neurosis."

These attacks, as I have elsewhere noted, come and go without apparent cause. They continue for a few days, and are characterized by nausea, uncontrollable vomiting, slight elevation of temperature and more or less pain in the head or in the gastric region.

The continued study of these cases has developed the fact that in a number of them there is a transient albuminuria, which continues during and for a short time after the other symptoms of the attack have disappeared. But before giving detailed attention to the albuminurias which accompany these lithemic attacks in early childhood, I wish to call attention to some clinical observations which I recently published in the *Philadelphia Medical Journal* on the association of transient albuminuria, arterio-sclerosis, and certain lithemic manifestations occurring in later life.

In this paper I noted the fact that migrainous and bilious attacks so common to lithemics in middle life and advanced life, are not uncommonly associated with a transient albuminuria, and that as time goes on there slowly develops in these individuals an arterio-sclerosis, and that as the arterio-sclerosis grows apace, the albuminuria accompanying the lithemic paroxysms increases, until finally it becomes constant. I further suggested that the treatment of this lithemic albuminuria should be preventative, that is to say, the treatment should be commenced either before the albuminuria appeared or that the importance of

the slight transient albuminuria occurring with lithemic headaches, and gastro-neurotic attacks, should be recognized and treated long before advanced arterio-sclerosis, and constant albuminuria had developed. And here again I raise my voice in evidence that the transient albuminurias of lithemic attacks are of the greatest pathological and clinical importance, in that they point out to us certain lines of treatment which must be persistently followed if these cases are to be saved in later life from cerebral hemorrhage and chronic Bright's disease.

The importance of this subject is increased by the fact that there is a long period of time covering late childhood and early middle life, during which lithemic paroxysms are as a rule not associated with albuminuria. I am impressed with the belief, therefore, that albuminuria accompanied by lithemic manifestations, is more frequent in early childhood and advanced life, than it is during late childhood and early middle life, that is to say it is less frequent in the middle than in the extremes of life.

As previously noted, one is able to account for the gradually increasing albuminuria of lithemic paroxysms in advanced life by the coincident gradual development of an arterio-sclerosis. It has its origin probably in the long continued irritation from toxic products which are the causes of these lithemic paroxysms. The transient but gradually increasing and finally continuous albuminurias of lithemic patients in advanced life has, therefore, in the gradually developing arterio-sclerosis with the accompanying changes which occur in the kidney, a sufficient morphological explanation, which does not apply to the albuminurias which accompany the lithemic manifestations of early life. For an explanation of the lithemic albuminurias which occur at this time, other etiological factors must be considered. With this somewhat general discussion of the subject, I ask attention to the following case which may serve as a text for the discussion of the etiological and the pathological importance of albuminuria as a symptom of early lithemic manifestations.

CASE I.—Male child, two years of age, born of gouty parents, history on both sides of bilious attacks, migraine, and rheumatic gout running through several generations. On March 8th, last year, I saw this child in the beginning of one of his severe lithemic attacks. I learned from the mother that he had had five or six of these attacks within a year and that they developed without apparent cause, continuing for four or five days, and con-

valescence was then rapidly established, the child remaining well until the next attack. The similarity of these "spells" had impressed the mother so that she spoke of the attack in which I saw him for the first time, as being the beginning of one of the peculiar attacks from which he so often suffered. The patient in this instance was prostrated with vomiting, which continued for four days and then disappeared as suddenly as it came. The patient had a temperature at this time ranging from normal to 102°, and was in a state of constant fretfulness, crying a great portion of the time. He was constipated, and toward the close of the attack when his constitution was relieved by small doses of calomel, the fecal discharges were very putrid. During the whole of this time the nausea was great. All food, even water, was quickly rejected by the stomach. On the fifth day, convalescence commenced, and was soon rapidly established. All symptoms quickly disappeared, and the child commenced to take and relish food, as he did when in perfect health. The urine of this child was carefully examined and found to contain albumin, hyaline casts, epithelial cells. Immediately convalescence was established the urine cleared.

The transient albuminuria which accompanied this attack is a point of special interest in this history to which I wish to call attention, but, in the first place, I wish to say a word as to the nature of this attack and to insist that it was primarily of lithæmic origin, and not a case of primary gastric or intestinal infection. These cases belong to the class of cases described by Holt under the name of "Cyclic Vomiting," and to the class of cases which I have previously described at length under the term "Lithæmic Gastric Neurosis." As I said in a paper read before this society last year, I have followed a number of these cases long enough to see these gastric attacks of infancy gradually change into true migrainous attacks in childhood.

As the child gets older there is a tendency to abatement on the part of the gastric symptoms, and an increase in the headache and narcotism which is more characteristic of them in later childhood and adult life. This gradual transition seems to stamp these attacks as purely of lithæmic origin. That these lithæmic attacks of infancy and early childhood may be accompanied by transient albuminuria, and other evidences of intense renal irritation is of more than passing importance and of especial interest

to pediatricists, since it cannot be explained by arterial changes at this early period of life.

The albuminuria, therefore, it seems, can only be due to the irritation of the delicate kidney structures of the child, which results from the attempt at elimination from the blood of the poisonous and irritating products which are the causes of the lithemic attacks. Since my attention has been directed to the subject, I have found not infrequently a small quantity of albumin in the urine of infants and children suffering from attacks of acute lithemia.

In this connection it is interesting to note that Lacour reports three cases of intermittent albuminuria which occurred in the same family, and called special attention to the fact that these children had inherited a strong gouty tendency.

It is my belief that these cases, which I have described, do not belong to precisely the same type of cases which Jacobi has so exhaustively described in his teaching over a period of many years, as occurring in the early days of infancy as the result of uric acid infarction, nor do they belong, as I believe, to the albuminurias which Jacobi has described as being produced by the absorption of toxic products which result from abnormal intestinal fermentations, occurring in the intestinal disorders of infants and children. But they are, on the other hand, as I believe, analogous to the transient albuminurias which occur as a result of lithemic paroxysms in later life. Auto-intoxication is responsible for lithemic albuminuria, whether it occurs at the beginning, or at the end of life, and that its prevalence in middle and later life is due to the arterio-sclerosis which this same auto-intoxication has developed, and that its prevalence early in life is due to the fact that the kidney at this time is more delicate of structure, and more non-resistant than it is later. The comparative infrequency of lithemic albuminuria in late childhood and early adult life, is due on the one hand to the better developed and more resisting structure of the kidney, and on the other to the fact that the arterial changes found in old lithemics have not yet had time to develop.

DEATH FROM HÆMOPTYSIS OF A CHILD AGED THREE YEARS.

BY GEO. N. ACKER, A.M., M.D.,

Clinical Professor of Medicine and Diseases of Children, Columbian
University; Visiting Physician to the Children's Hospital and
to the Garfield Hospital, Washington, D. C.

C. P., aged three years, male, colored, was admitted to the Children's Hospital April 20, 1898.

Mother and father living and in good health. One sister, eighteen months old in good health. Father has lost two brothers with tuberculosis. The child was breast fed until two months old and cut first teeth when six months old. Has always been a delicate child. He has been sick for twelve days with a slight cough which has increased, until at present it is frequent and attended with much pain. He has had fever every night since the sickness began.

There is loss of appetite and he vomits frequently after eating. The bowels are irregular. He is very restless at night. The cervical glands are somewhat enlarged.

There is slight dulness on percussion on both sides of chest. Rough broncho-vesicular breathing, with many mucous râles both sides of chest, more marked on the right side.

The child is very nervous and fretful. The patient became progressively worse and he died April 29th at eleven a.m., immediately after expectorating a quantity of blood. The temperature at the time of death was 104° F.

The temperature ranged during the time the child was in the hospital from 100° F. in the morning, to 104° F. in the evening.

Necropsy by Dr. D. S. Lamb.—Body somewhat emaciated; follicular enlargement of tonsils; slight abrasion of right vocal cord; otherwise mouth and larynx normal, some thin blood in mouth, pharynx, larynx, and trachea.

Both lungs congested and studded with miliary tubercles; œdema of right lower lobes; some bronchitic exudate on right side; bronchial glands much enlarged and cheesy; bloody serum in each plura; heart normal; liver pale; some bile in gall bladder;

enlarged gland at head of gall bladder; spleen much enlarged; firm old adhesions to diaphragm; congested; studded with either enlarged malpighian bodies or minute tubercles throughout; pancreas normal; stomach normal; intestines normal; mesenteric glands enlarged and cheesy; each kidney contained a few tubercles; cause of death, apparently from hæmoptysis; no ulceration.

913 SIXTEENTH STREET.

THE URINE OF HEALTHY INFANTS AND CHILDREN.

BY FRANK SPOONER CHURCHILL, M.D.,

Associate in Diseases of Children, Rush Medical College, in Affiliation with
the University of Chicago ; Professor of Pediatrics, Chicago Polyclinic.

The factor of excretion in the human economy is such an important one, and the well-being of the subject depends to such an extent upon its proper performance, that the kidneys through which, together with the intestinal tract, it is chiefly accomplished, have received much careful study—at least in adults. Comparatively speaking, however, little exhaustive work on the urine of children has been done, and when, in the routine analysis of urine from his patients one seeks for a normal standard, he finds but little physiological with which to compare his specimens pathological, and has difficulty in determining to what extent proper renal excretion is going on, certain only that in this direction something is wrong, though exactly what he is unable to determine.

I have in my own practice felt the need of a normal physiological standard of the urine of children with which to compare the urine of abnormal cases, especially those more or less obscure, and I have been investigating the urine of a few healthy infants and children, and now lay the result of the work before the Society for discussion and as a contribution for future reference.

The subject seems to me an important one. While we do not necessarily detect actual present organic disease of the kidney by careful analysis of the urine in all our patients, will not such analysis often clear up obscure and doubtful cases? Will it not give us valuable information as to the extent to which metabolic activity is proceeding? May not the study of many such analyses possibly reveal tendencies latent in early life, but subsequently developing into apparent and actual disease? And may we not from this study possibly glean information of value to us, in seeking to check these tendencies, in helping our patients overcome what is so often an hereditary handicap?

I have made no attempt in this paper to discuss the urine from abnormal and unhealthy children, but have limited myself strictly to the healthy. My investigations have been made upon

seventy infants and children ranging in age from one day to twelve years, forty-eight girls and twenty-two boys, and the results classified as to age and averaged. One hundred and forty-six specimens have been analyzed, it being better to examine two or more samples from a limited number of cases than a single specimen from a larger number of children. The results have justified this method of procedure, as considerable variations in the amount of urine passed at different times have been noticed.

Observations have been made upon the following points: Total amount of urine in twenty-four hours, color, reaction, specific gravity, presence or absence of albumin and sugar, percentage and total urea, percentages of the chlorides, phosphates and sulphates. The sediment was examined macroscopically and microscopically. The ferrocyanide and nitric acid tests were employed for the albumin, the urea was estimated by the hypobromite method—Doremus' ureometer being used; the percentage of the chlorides, phosphates and sulphates determined by centrifugal analysis, as recommended by Purdy, the tubes being revolved three minutes, at a speed of 1100 revolutions per minute.

The urine was obtained chiefly from children of the Chicago Half-Orphan Asylum, and, notwithstanding the efficient care and co-operation of the nurses and assistants, it was, of course, impossible to collect accurately the total twenty-four hour amount in all cases attempted. Specimens about which there was any doubt were rejected, and the averages are based only on those known absolutely to be correct.

Examination in detail of the results as given in the tables shows several interesting points, which will be briefly considered.

AMOUNT.—The daily amount of urine passed by my cases is much less than that recorded by most authors whom I have been able to consult, except Herz, whose analysis upon sixty cases—thirty girls and thirty boys, between six and fourteen years of age—correspond approximately with my own. A comparison of my results with those quoted by Rotch and Holt, however, shows a marked discrepancy, for which I am unable to account. I should be suspicious that I had not been successful in getting the whole amount in my cases had I not taken especial care in this direction. Moreover, the specific gravity confirms the amount.

SPECIFIC GRAVITY.—We see here a higher average than is given by the authors already quoted, a condition we should naturally expect in the urine of children passing but a small amount. Had we failed to collect the total amount, we should expect a lower specific gravity than is recorded, inasmuch as even those cases which have been thrown out had a fair admixture of night and day urine.

Reliable as are the observers quoted, should we not expect to find a comparatively high specific gravity in the urine of children of this age, at a period of great physical activity with consequently greater elimination of urea? I shall recur to this point below in speaking of the latter element.

The specific gravity of the one young infant which I have been able to record is low, coinciding with the well-known observation at this period; it ranged from 1001 to 1005 from the twelfth day to four weeks. It is, however, generally higher during the first two days of life, before the establishment of the breast milk. It drops after this, and continues low throughout the first year, owing to the fluid character of the infant's food. During the second year, solid food being added to the diet, the specific gravity rises; and in four cases, aged respectively twelve, thirteen, eighteen and twenty months, I found it ranging from 1026 to 1030, the urine being a mixture of the day and night eliminations.

UREA.—The estimation of this constituent is perhaps the most important of all the urinary solids, being as it is an index of general metabolic activity. As we should expect from their greater activity, and as Purdy and Foster state, we find the urea excretion in children relatively higher than that in adults. The low percentage noted during early infancy is, of course, due to the quiescent state of the child. Martin and Ruge, however, report wide variations in single specimens during the first ten days of life, ranging from 6 per cent. to 1.9 per cent. Schiff also gives wide variations, placing averages at from .28 per cent. to 1.7 per cent. during the first fourteen days. Why there should be such a wide range in the excretion of this substance at a time of such quiescence, it is difficult to see. Possibly greater metabolic activity after nursing may account for it. I have no statistics upon the relative amount of urea in urine passed just before, just after and some time after feeding. The few observations I have made at this age showed, without exception, very low percentages, from

Age.	Weight.	Sex.	Amount in Single Specimen.	24 Hour Amount.	Specific Gravity.	Urea. <small>PER CENT.</small>	Chlorides. <small>PER CENT.</small>	Phos- phates.	Sulphates.	REMARKS.
1 day	3.18 kil.	F	3 c.c.			.4				
12 days	3.86 K.		36 c.c.		1003	.1			Passed at 12 M.	
3 wks.	4.14 K. One				1001	.5			" " 9 A.M.	
3½ "	4.25 Infant	M	30 c.c.		1005	.2			" " 4 P.M.	1 hour after breast
4 "	4.47		23 c.c.		1004	.1				
1 year	9.98		9 c.c.		1021	2.6				
1 "					1026	2.7	12 <small>PER CENT.</small>	16 <small>PER CENT.</small>	1.75 <small>PER CENT.</small>	Dentition slow.
1½ yrs.		F		195 c.c.	1030	2.5	18 "	11 "	1.25 "	1 Specimen
1 3 mos.	9.76 kil. (21½ lbs.)	F			1026	2.3	11 "	11 "	1.00 "	Mixture night and day urine
20 "				290 c.c. +	1028	2.8	16 "	13 "	1.75 "	
20½ "		M		325 c.c. +	1026	2.1	8 "	12 "	1.00 "	
2 yrs.	14.07 K. (31 lbs.)	M		450 c.c.	1020	1.7	6.5 "	6 "	1 "	
		{ 3 M } { 3 F }	12 Specimens in all.							

1.20 per cent to .4 per cent.—lower than those cited. After the first year it rises, and from three to twelve years my 133 specimens show a higher general average than that usually given. I can but think Verordt's percentage, based on only seven cases, is too low. He records it at 1.1 per cent. to 2 per cent., four being below 2 per cent., one 2 per cent., and one 2.6 per cent. and one not given. This represents the adult average, whereas, so great is the physical activity of the growing child, so active is his metabolism, that a large amount of urea is formed, and while it may be argued that most of his nitrogenous food goes to the building up of the rapidly growing body, and thus the amount of urea formed in the urine would naturally be less, it would seem more rational to expect a greater elimination of this substance. Not only are my average percentages higher than the average given for adults, but individual cases show a remarkably high percentage of elimination of urea, eight children having over 3 per cent, the highest being 3.7 per cent.

The amount of urea per kilogram of body weight, while slightly higher than the ratio given for adults, is lower than that given by other observers, as we should expect from the smaller amount of urine.

CHLORIDES.—The chlorides were found quite constant at about 11 per cent. up to seven years, after which they were about 9 per cent.

PHOSPHATES.—The phosphates were found to be from 8 to 11 per cent. from 3 to 5 years; 5 to 7 per cent. from six to twelve years—the adult range being about 8 per cent. It has been suggested that the smaller amount of phosphates found in the urine of children, is due to the fact of the phosphoric acid being retained in the body for the growth of bone. One specimen from a year old boy showed 16 per cent., and as he was somewhat slow about teething, though otherwise perfectly healthy, the question suggested itself as to whether substances which normally go to build up the teeth, were being eliminated as phosphates, and if so, why; digestion was absolutely normal. No conclusions, however, can be drawn from one solitary instance; the observation is merely of speculative interest.

SULPHATES.—The percentage of sulphates was 1 to 1.2 per cent., slightly higher than in adults—.8 being their average. Purdy states that the sulphates run parallel with the urea.

ALBUMIN AND SUGAR.—Neither albumin nor sugar were de-

tected in any specimen. So much had been said about a physiological albuminuria that I had expected to find albumin in one or more specimens. It must be remembered, however, that my cases were examined but two or three times, and some only once, and that therefore a transient temporary albuminuria might have come and gone between examinations. No deductions can be made on this point.

SEDIMENT.—Examinations of the sediment showed nothing of especial interest in any case.

REACTION.—The reaction was acid in all cases, though of course varying in intensity in different specimens.

COLOR.—The color in most cases was pale, in the rest normal.

Looked at as a whole the records show three factors of chief importance. The small amount of urine, the high percentage of urea, and, a natural result of these two, a high specific gravity. In other words, these children are passing a comparatively concentrated urine. They are all healthy, robust children, eating, sleeping and digesting well, and of average weight. Do these records of their urine represent the urine of average American children, or of average children living under American customs and régime? Does the difference in nationality account for the difference in results as obtained by investigators cited, and by myself? Their cases were all German children, mine American, though mostly of foreign parentage and living in an asylum. Or, is it merely a coincidence, happening among this small number of children, that they all pass a urine, small in amount, concentrated in character? The number of cases is too small to draw conclusions as to the effect of race, national habits and customs of life.

It is interesting to note, however, in this connection the amount of urine in adults, as given by observers in different countries. Simon gives the following:

Salkowsky (Germany),	-	-	1500 to 1700 c. c.
Jaksch (Austria),	-	-	1500 to 2000 c. c.
Landois & Sterling (England),	-	-	1000 to 1500 c. c.
Gautier (France),	-	-	1250 to 1300 c. c.
Simon (America),	-	-	1000 to 1300 c. c.
		(Females),	900 to 1000 c. c.

It would seem from these tables that American adults and also children, pass smaller amounts of urine than the people of other countries.

Are we not justified in regarding these facts as unfortunate and the development of renal trouble later in life, as a not at all improbable result of the constant irritation to the kidney? Is it not advisable, especially in children with a renal, rheumatic or lithæmic heredity to inquire carefully into the amount and quality of their food and drink, and to take measures to insure the elimination of a more dilute and less irritating urine? This would seem to be all the more important when we consider the large number of conditions which act, either directly and immediately, or indirectly and remotely, as causes of nephritis, a disease occurring in childhood more frequently than is generally thought. We must seek to correct or remove any condition of the system which might act, however slightly, as an irritant of the kidney.

As stated in the beginning of the paper, the subject of renal excretion is an important one, and the questions asked are put with the hope of a full discussion and to gain further information.

I wish to express my indebtedness to Dr. Charles W. Purdy for valuable suggestions in the preparation of this paper, and also to Dr. Wesley Thomas for the free use of the wards at the Chicago Half-Orphan Asylum.

583 EAST DIVISION STREET.

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DISCUSSION.

DR. CHRISTOPHER.—I am much interested in the last column in the table presented by Dr. Churchill. He gives there the urea per kilogram. I am in the habit of estimating that the amount per kilogram is 0.5 gramme, or that it is $\frac{1}{2000}$ th part of the weight of the individual. It is remarkable how near his figures come to that ratio. If one to two thousand is the correct ratio, then the number in the last column should be .500 to bear it out, and it runs almost universally .600, except at seven years of age, where it is down to .296, about one-half the quantity at every other age. I beg to call especial attention to that apparently abnormal figure. It is an entirely new one to me, and I think it has a bearing on the paper I shall present to-morrow.

DR. TYSON (an invited guest).—A good deal of the matter of the paper is quite new to me, as far as the discussion of details of the different proportions of the constituents of the urine is concerned. I have myself made no practical study of this subject, but there are two points that have occurred to me, in connection with which I have had some experience. The first is the question of albuminuria of children. In my experience the albuminuria of children is not found at as early an age as any of the ages here recorded. I think commonly twelve to fourteen years is about the age at which this albuminuria presents itself, and it is apt to last until twenty-one or twenty-two years of age, when the condition disappears. The second point, in regard to which I have had some little practical experience, is the quantity in adults. My experience with the quantity of urine in adults corresponds with that of other American observers. I think it is more often below 1500 c.c. than above it; in fact, 1200 to 1500 c.c. is what I would report from the observations I have made.

DR. FRUITNIGHT.—I would like to know whether any consideration of the compensating action of the skin was taken in regard to the season of the year.

DR. CHURCHILL.—The observations were begun in September and carried on until the last of March.

DR. ROTCH.—This is a very valuable work, and a work which is needed. When I was preparing the tables to which reference has been made, I had great difficulty in getting any information I could rely upon. I have no doubt that this work of Dr. Churchill is a valuable supplement.

DR. GRIFFITH.—I would like to know if there was any limitation in the amount of water these children could have. For instance, it is possible that in a public institution water might not be as freely supplied as in a private family where a child can have water whenever it desires.

DR. CHURCHILL.—I inquired that of the attendants and they said the children were allowed water whenever they wished it, and as far as they observed they drank as much as other children.

DR. HOLT.—How much milk was allowed the children?

DR. CHURCHILL.—As much as they liked to drink. In answer to the question in reference to the albumin where I found casts, there was no albumin in any of the cases except in the child that developed an attack of acute nephritis a month after my first examination. When I came to collect the urine a second time he was in the midst of an attack of nephritis which had been diagnosticated as an attack of "cold" by the homeopathic physician. The specific gravity was always taken from the twenty-four hour urine.

SHORT REPORTS ON UNUSUAL CASES.

BY T. M. ROTCH, M.D.,

Professor of the Diseases of Children, Harvard University, Boston.

CEREBRO-SPINAL MENINGITIS.

On January 17th, during my service at the Children's Hospital, a little girl three and a half years old entered my wards. The history of her sickness previous to entering the Hospital was such that the diagnosis of no especial disease could be made. She had had a little vomiting and headache, but nothing definite could be ascertained. The physical examination, with the exception of showing that there was rhachitis, presented nothing abnormal. The temperature ranged from 98.5° to 105° F., the pulse from 98 to 140, and the respirations were almost invariably 30.

An examination of the blood by Dr. Wentworth about six weeks after onset, showed no marked changes in the size or form of the red corpuscles, and was as follows:

Red corpuscles, - - - -	2,800,000
White " - - - -	11,200
Hæmoglobin, - - - -	40 per cent.

The differential count showed

Neutrophiles, - - - -	72 per cent.
Small mononuclears, - - - -	10 " "
Large " - - - -	17. $\frac{1}{3}$ " "
Eosinophiles, - - - -	0. $\frac{2}{3}$ " "

No plasmodia were detected.

A lumbar puncture was then made by Dr. Wentworth, the diplococcus intracellularis was found, and the diagnosis of chronic cerebro-spinal meningitis made.

The subsequent course of the case pointed decidedly towards this disease, which lasted from February 24th to April 22d. I report it to show the value of lumbar puncture in cases where the diagnosis is obscure. At the time the lumbar puncture was made the child seemed to be failing fast, but soon began to improve after a number of remissions of the symptoms which are so characteristic of the chronic form of cerebro-spinal meningitis in children.

The accompanying chart shows the temperature at a time when nothing especially characteristic of the disease could be detected from it.

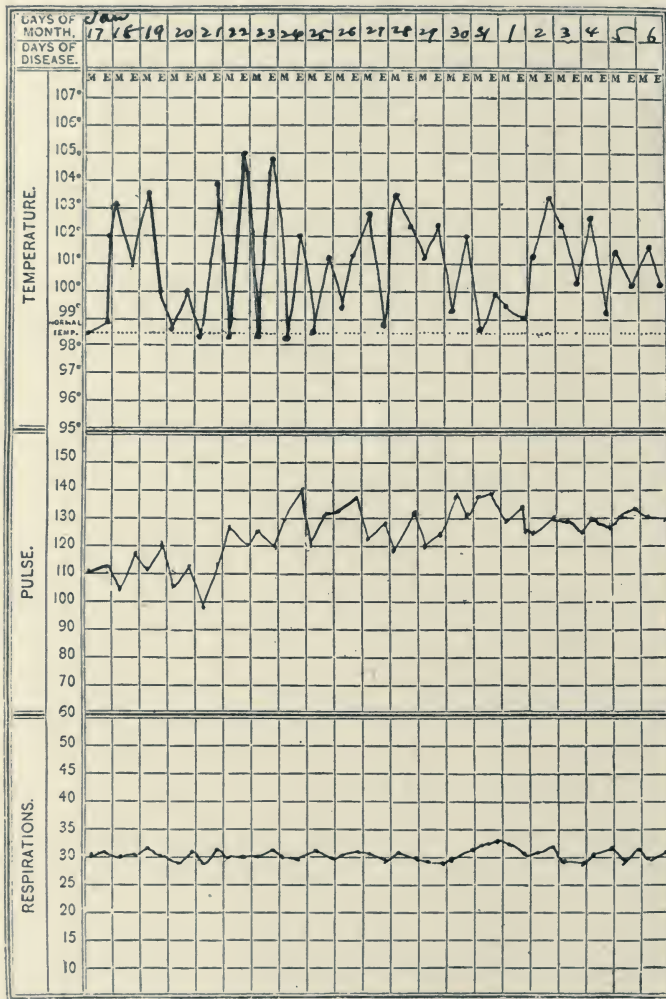


CHART SHOWING RANGE OF TEMPERATURE, PULSE, AND RESPIRATION IN A CASE OF CEREBRO-SPINAL MENINGITIS BEFORE CHARACTERISTIC SYMPTOMS DEVELOPED.

Published by CODMAN & SHURTLEFF, Boston.

INTUSSUSCEPTION.

On the 9th of November, 1897, I saw, in consultation with Dr. Thompson, of Boston, a girl five years old with a history of

previous good health, except that six months before she had had attacks of paroxysmal abdominal pain and vomiting, from which she completely recovered. On November 6th she complained of abdominal pain, which increased during the night, and early in the morning she began to vomit. I saw her twenty-four hours after she had been taken sick. She had not passed any fecal matter from the bowels for twenty-four hours, but only bright red blood mixed with mucus. The pulse varied from 90 to 99 and was weak. The temperature was 100° F.

An examination of the abdomen showed a mass in the right hypochondrium which was dull on percussion. A rectal examination failed to reach the tumor. The child's face was pinched and she was evidently growing worse rapidly. While waiting for the preparations for laparotomy to be made I tried hydrostatic pressure, by means of a fountain syringe with the usual nozzle, at a height of about four feet, and almost instantly the tumor disappeared, the child's face lost its anxious expression, and from that time there were no more symptoms of intussusception.

I merely mention this case as showing how under very rare circumstances a pronounced intussusception can be reduced with very slight pressure, provided, of course, as was probable in this case, the axis of the invaginated portion was in a direct line and no adhesions had taken place.

The next case was that of a boy two years old who entered my wards at the Children's Hospital with the following history: He had previously had no diseases and had no history of not being well and strong up to the time of entrance. Twelve days before entering the hospital he fell out of bed and on the following day vomited several times. Since that time he vomited nearly all the food that was given to him and had profuse diarrhoea consisting of light yellow, watery movements. On physical examination his eyes were found to be sunken, and he seemed very sick, but his pulse was full and regular and nothing abnormal was detected in the heart, lungs, or abdomen. During the following week he improved rapidly, sat up, laughed, played and was apparently perfectly well, except that after each movement of the bowels he passed a small quantity of bloody mucus. The movements, however, were well formed and yellowish brown. A lumbar puncture made four days after entrance, on

account of an irregularity of the pupils which was detected at the time, showed a normal fluid.

Eleven days after entrance he began to be restless, vomited in the evening and cried a great deal during the night. On the following day he vomited all the food that was given to him and had no movement of the bowels excepting a little blood and mucus. At 8 o'clock in the evening a tumor was detected in the left iliac region, and at 9:30 it had increased in size, extending down the whole side of the abdomen to the anterior superior spine of the ilium. The child was looking very badly, had a rapid pulse, sighing respirations, and waxy color. The abdomen was greatly distended, and the coils of intestine showed plainly through the abdominal walls. There was continued tenesmus and passage of blood and mucus. At 10:30 P.M. hydrostatic pressure at a height of five feet, by means of a fountain syringe, was tried. The tumor immediately disappeared and the child seemed in good condition. At 10 o'clock on the following day no tumor could be found, but two and a half hours later a mass could easily be felt in the same location as before, but not so large. Hydrostatic pressure again caused the disappearance of the tumor, but the same conditions returned at 3 P.M., and the child was looking so badly that he was transferred at once to the surgical side of the hospital, where he was operated on two hours later by Dr. Lovett. An intussusception of the ileum into the cæcum was found which had apparently existed for a long time, as the layers of intestine were firmly adherent. There was also a smaller intussusception apparently recent which was easily reduced. The larger one resisted all attempts at reduction and was accordingly resected and an artificial anus formed. The child died four hours later.

I report this case as illustrating the possibility of an old and a fresh intussusception occurring in combination, and in order that we should avoid giving a too favorable prognosis when a recent intussusception has been reduced.

197 COMMONWEALTH AVENUE.

DISCUSSION.

DR. HUBER.—The two cases of intussusception reported have interested me considerably. Since October I have seen six cases; two, with tumors, were reduced with the fountain syringe. Two others have passed from under my observation; two seen within

the past few months have interested me particularly. One was seventeen weeks old, and the child was seen about twelve hours after the initial symptoms. A careful examination failed to show any tumor. A high rectal enema under moderate pressure allowed a good deal of water to enter, showing that the seat of the trouble was high up. The water escaped, but the symptoms did not improve. The child was sent to the hospital, a laparotomy done, and an intussusception three inches in length found at the ileo-cæcal valve. The little one recovered from the immediate effects of the operation, but died six weeks later of some secondary cause. The second case, a child of five months, was seen about nineteen hours after the initial symptoms. A particular point I wish to refer to is the slight amount of blood that is sufficient to warrant the diagnosis of this condition. A careful examination under chloroform failed to show any tumor, yet on laparotomy we found that the intussusception started at the ileo-cæcal valve and extended along the ascending and transverse colon into the descending colon. Yet careful palpation failed to reveal any tumor. There was no question as to the diagnosis in these cases, because at laparotomy the condition was established. The child was rachitic, had tetany to a marked degree, ran along about five weeks, no attempt on part of wound at union, and the patient simply died of exhaustion.

DR. FRUITNIGHT.—Dr. Rotch's remarks are very appropriate, for we are prone to neglect measures other than purely operative. Last fall I saw a child about three years old in whom a diagnosis of intussusception had been made by several able physicians. An operation was made, and when the abdomen was opened it was found that the indications of intussusception were there, but it had become spontaneously reduced. The hydrostatic pressure in Dr. Rotch's case fortunately saved the child an operation. In another case, in which there was tenesmus with bloody discharge, the child was also relieved by this method of treatment. It will be remembered that very often on the *post-mortem* table intussusception is frequently discovered in the bodies of young children, which were unaccompanied by any clinical symptoms. At times more than one such lesion has been found in the same subject. It is plausible to assume that such cases of intussusception as these often reduce themselves spontaneously, the lesion having gone undiscovered because no symptoms had been developed.

DR. HUBER.—I would like the doctor to understand that in the cases reported the high rectal enema had been tried, and as the symptoms did not disappear operation was resorted to.

DR. WINTERS.—I wish to recall a case which I saw in consultation with Dr. Eastman. We made a diagnosis of intussusception and sent out the father to telephone for a surgeon to

operate. During his absence distention was tried with complete relief and no operation was necessary. In another case in which we tried hydrostatic pressure relief was immediately obtained. That was in the morning about ten o'clock. That evening symptoms of the return of the intussusception occurred; the child went into a collapse and died. So it seems there is some doubt as to the permanency of the result. In that instance the second intussusception recurred about eleven hours after the relief of the first, and the child died in two or three hours.

DR. NORTHRUP.—I wish to endorse what Dr. Huber has said in regard to the diagnosis. We all meet individual cases and we all find some that can be reduced by injections, some which cannot, and some which are harmed by them. I recently met an individual who was elated over his success in a case where he took the fountain syringe up a flight of stairs, the patient remaining in the lower hall, but he did not succeed; so he carried it up another flight and reduced the intussusception. I would like to hear whether we would endorse any certain height. I have met a case which would not go back with gentle injection, neither when the abdomen was open was it safe to use sufficient traction to reduce it.

DR. CONNER (By invitation).—Cases of intussusception are met not infrequently, as has been said. Some are reducible without difficulty, some reducible with difficulty and some are absolutely irreducible. It has been my fortune to see a few of these cases, not very many, and I can heartily endorse what has been said about the propriety of what has been adopted. Special care should be observed in the use of these injections or far more harm may be done than good. We are frequently called upon to operate after harm has been done in this way, and the individual is far worse off than if nothing had been done.

DR. JACOBI.—Only as to the height of the water. There was a medical gentleman in New York who insisted upon having the water run through a tube exactly 14 feet long, no less and no more. Now the use of a tube 14 feet long requires just exactly what Dr. Northrup says, taking the syringe upstairs. The gentleman mistook a baby's bowels for iron pipes, and tried to exactly calculate the hydrostatic pressure so that 14 feet was just the proper thing. That is so unphysiological and so opposed to everything we know of the infant gut, its tissues, its expansibility, its power of resistance, etc., that it is hardly worth while to consider that barbarous procedure. If an injection is to be made it should be made from a height of a foot or a foot and a half, and no more. That, with a little massage, while the hips are raised and the baby on his face, will accomplish everything that is to be accomplished. A thing not to be forgotten is that the intestine was not previously in a normal condition.

Sometimes there is a local peritonitis, and I have seen a few instances where perforation of the gut took place from overloading, or from trying mechanical (sound) reduction.

DR. CAILLÉ.—In those cases with tumors presenting, I think the best rule is the one we have in Dr. Holt's book. One prolonged attempt of reduction under anæsthesia should be made by inflation, and then an operation should be resorted to if we are unsuccessful.

DR. ROTCHE.—I did not advocate this method of reducing intussusception, in fact I expected exception to be taken to it. We should adopt this method of reducing an intussusception with the greatest care. The surgical opinion, I think, is against using it at all. In some cases we undoubtedly cause death by rupture. However, the opinions of those who are not surgical are not quite so strong as of those who are purely surgical. If the intussusception cannot be reduced by slight pressure, increased pressure should not be used, because then adhesions have occurred. I happened to have a case where the axes of the two pieces of intestine were the same, otherwise they would have been pressed still more tightly together. Therefore I should say that a very slight pressure for a very short time only should be used. The infant may appear perfectly well; again, no tumor may be felt, and yet the other symptoms be so significant that laparotomy should be done at once.

CONGENITAL MALFORMATION OF THE BILE DUCT.

BY CHARLES P. PUTNAM, M.D.,

Boston.

The case was referred to me by Dr. W. W. Miner of Ware, and was also examined by Dr. J. W. Elliot, who operated, and Dr. T. M. Rotch.

The patient was a girl, four months old, of healthy parentage, and free from evil pre-natal influences as far as is known, (except that the mother had had an attack of something like dysentery six months before the child was born).

The birth weight was six pounds eight ounces, and up to the age of two months and a half the child gained weight every week, and at that age weighed seven pounds, fifteen ounces. Since then it had gained irregularly, but at four months weighed nine pounds thirteen ounces.

It had been on the breast four weeks, and after that had had various mixtures.

The skin became yellow two or three days after birth, and had been yellow ever since, in varying degrees.

The meconium had been black, but scanty, only two or three discharges. All subsequent feces had been quite or nearly white, very little change of color having been brought about by changes of food, and none by calomel or podophyllin.

Vomiting and diarrhœa had occurred occasionally, but had never been excessive, nor were there other marked signs of indigestion.

Nevertheless the child had cried a great deal as if in distress, especially at night, and had been quieted only with difficulty.

As for the effect of foods, apparently condensed milk much diluted had suited the digestion best, and conversely all the preparations containing more fat had disturbed the digestive organs more.

The liver was large and hard, and was easily felt about an inch below the edge of the ribs. Otherwise the appearance was that of a small but fairly well-looking child, somewhat yellow, but nevertheless somewhat pink on various parts of the body. It had not the general appearance of a child affected by a grave organic disease.

The white feces were carefully subjected to two delicate and intricate tests for bile elements by Dr. C. R. L. Putnam with negative results.

The published cases of this affection show that it has been fatal without exception, not plainly, because the bile is not poured into the intestine, but because the bile retention gives rise to cirrhosis. Bile can be supplied to the intestine artificially, but there is no way to prevent the cirrhosis.

The operation was decided on therefore for the following reasons:

- (1) Because the child was sure to die without it.
- (2) Because it might prove possible to carry the stream of bile into the intestine.
- (3) Because, failing of this, it might be possible to make a biliary fistula and relieve the liver, supply bile artificially and keep the child alive, until it should be large enough for a more extensive operation.

During the few days while the operation was under consideration the child was evidently failing, in spite of a careful supervision of its food.

The child was placed in the inversed Trendelenburg position. An incision having been made in the median line, and the air having been admitted, the intestines fell away and it was easy to see the lower surface of the liver. The gall bladder was in its proper place but was of a pinkish color, and evidently contained no bile. On pushing it to the (child's) left, a duct filled with bile came into view. This was emptied temporarily by stroking it to the right or toward the liver, but became distended on being stroked in the opposite direction, showing that it was obstructed on that side. The probe also passed to the right but not to the left. As no way presented itself of bringing about a normal condition, a small glass tube was introduced to drain this duct and the wound was closed.

A considerable quantity of bile ran out through the tube, and the child passed the quietest night of its life. Next day it began to fail, however. A slightly elevated was followed by a normal and subnormal temperature, and it died on the third day.

THE AUTOPSY showed normal conditions except for the organs described as follows by Dr. W. F. Whitney:

“The liver and a portion of the duodenum and stomach were examined after hardening in alcohol.

“The relations at first sight appeared normal. The liver was, however, coarsely granular on the surface with alternating slightly opaque, prominent areas separated by more translucent fibrous looking ones of a dark green tinge. The organ measured 11.5 by 6 by 3 cms., and the right and left lobe were distinctly marked, while the lobe of Spigelius and the quadrate lobe were hardly indicated. On dissection at the portal fissure was a little dark green connective tissue, but not the slightest trace of a duct, although the vessels were normal in size and relation. The gall bladder lay in its fissure and a duct could be distinctly traced from it to the papilla in the duodenum where it opened.

“A section through the liver showed the bile ducts distended and filled with dark green inspissated bile. These could be distinctly followed to the green stained connective tissue first mentioned. The section surface showed everywhere irregular fibrous strings separating lobules of liver substance.

“Microscopic examination showed the fibrous tissue strings to contain vessels with thick walls and very numerous bile ducts of small size. The connective tissue itself contained relatively few nuclei and the line between it and the liver substance was quite sharp and the liver cells well preserved close up to it. In places it could be seen working its way in between the cells and evidently causing their atrophy. Small bile radicles filled with bile were occasionally to be met with among the cells.

“The condition is one of a failure of development (agenesis) of the hepatic duct, with bile retention, connective tissue growth with proliferation of the bile ducts and atrophy of the liver cells.”

Of course it was recognized that this operation offered but a very minute chance of success. For one thing it came later than it ought to have come, when the liver was already cirrhotic. We believe, however, that the right principle for the first time guided the treatment, in that cirrhosis, and not lack of bile, is the essential danger of the affection, and that on some other occasion an operation may be successful which diverts the bile out of the liver, even if it is impossible to turn it into the intestine.

SEVEN CASES OF LARYNGEAL DIPHThERIA TREATED WITH ANTITOXIN. ONE DEATH.

BY CHARLES GILMORE KERLEY, M.D.,

Lecturer on Diseases of Children, New York Polyclinic; Assistant Attending
Physician, Babies' Hospital; Attending Physician Out-patient
Department, Babies' Hospital.

The following cases of laryngeal diphtheria comprise the entire number treated by the writer with antitoxin.

The first patient was a boy, three and one-half years old. He was fairly vigorous; had been ill three days when first seen. I was called on account of croup, which failed to respond to home remedies. I found the patient sitting up in bed playing. There was slight dyspnœa, almost complete loss of voice, a hard, brassy cough. There was moderate glandular swelling at the angle of the jaw, no visible membranes, fauces congested.

TREATMENT.—Laxatives, liquid nourishment, calomel fumigations, ten grains every three hours. Culture taken.

The following day, the second under observation, I was hurriedly called and found the patient suffering from intense dyspnœa. Both inspiration and expiration were markedly impeded. Intubation was hastily performed with complete relief of the dyspnœa. One thousand units of antitoxin were given at once. Further treatment consisted of liquid nourishment, and strychnia sulphate, $\frac{1}{16}$ grains, six hours apart. Board of Health reported diphtheritic bacilli.

Third day under observation, there was no marked change in the patient. He breathed easily with tube. Moderate prostration. Temperature ranged between 100 and 101°. One thousand units of antitoxin was repeated.

Fourth day, patient much brighter, temperature normal, cough troublesome.

Fifth day, patient continues to improve, sat up in bed and played with his toys.

Sixth day, tube removed, but had to be replaced in thirty minutes on account of the return of dyspnœa. No other unfavorable symptoms developed.

Eighth day, tube again removed and not replaced. Patient made a good recovery. Developed a paralysis of the left leg

which lasted about three weeks. Throat free from bacilli in three weeks. Urine negative throughout illness.

The second patient was a girl ten years old; previous condition excellent. First seen on the third day of the illness. Temperature 103° , pulse 150, voice lost, hard, teasing cough, moderate dyspnoea. Throat filled with yellowish discharge. Both posterior pillars covered with grayish-white membrane. Posterior portion of uvula covered. Several small areas on pharyngeal wall, small thin layers on both tonsils. The examination of larynx unsatisfactory, but sufficient to show membrane on under surface of epiglottis. There was considerable swelling of the cervical glands. The examination of the urine showed a trace of albumin.

TREATMENT.—Two thousand units of antitoxin, $\frac{1}{150}$ grains of strychnia sulphate every six hours, steam spray, Dover's powder for cough and restlessness.

Second day under observation, temperature $101\frac{1}{2}^{\circ}$ to $102\frac{1}{2}^{\circ}$, pulse 120. Cough still troublesome, breathing easier, less dyspnoea. In general appearance the child was greatly improved and claimed that she felt much better. Throat examination showed one tonsil clear, membrane generally appeared less adherent. At four o'clock in the afternoon, a piece of membrane $\frac{1}{4}$ x 1 inch was coughed up from the larynx. Examination of the urine still showed a trace of albumin. Treatment—Strychnia, nourishment and steam inhalations as before. Irrigation of the nose and throat with a normal salt solution was carried out every three hours.

Third day, temperature 100° to 101° ; child showed marked general improvement, and asked for food. Membrane still on pillars, but the throat had a much clearer appearance. At seven o'clock in the evening another large piece of membrane was coughed up from the larynx.

Fourth day, throat clear of membrane; temperature 99° to 100° ; pulse 110; voice returned; coughed much less. Treatment as before.

Fifth day, voice improved, coughed very little; urine negative. Treatment unchanged.

Sixth day, voice normal.

On the eighth day under observation, the eleventh day of illness, the patient sat up in a chair apparently as well as ever. The throat was free from bacilli the thirteenth day of the illness. No sequela.

The third patient was a boy two years old, a strong and vigorous child. First seen on third day of illness. At first visit he was suffering from intense dyspnœa, both inspiration and expiration being impeded. Both tonsils and fauces covered with a dirty gray membrane. Sanious discharge from nose. Glands at angle of jaw moderately enlarged. Temperature 103°, pulse 150, fairly good. Child was intubated at once, with complete relief to the dyspnœa. Further treatment, stream spray, strychnia $\frac{1}{100}$ grains every six hours. Liquid nourishment. Antitoxin 2000 units. The following day, second under observation, child some brighter, membrane presented a broken down granular appearance; temperature 102°, pulse 150. Other treatment unchanged.

Third day, temperature 100°, pulse 120, membrane clearing, child apparently much better. Treatment unchanged.

Fourth day, throat clear, child playing with toys. The tube removed on seventh day. No return of dyspnœa, child made good recovery. Urine negative throughout. No sequela.

The fourth patient was a girl three years four months old. Previous condition strong, well-nourished. Child had been ailing slightly with croupy cough and hoarseness for several days, when suddenly at 8 A.M. she was taken with urgent dyspnœa. There were supra and infra sternal recessions. Both inspiration and expiration were impeded. Voice lost. Temperature 101°, pulse 130. Fauces generally congested with small spot of membrane on posterior pharyngeal wall the size of a flaxseed. Moderate glandular swellings at angle of jaw.

TREATMENT.—Liquid nourishment; emetic; laxative, calomel fumigations, ten grains every three hours. Intubation seriously considered. Culture taken. In the evening temperature 102°, pulse 160, very soft and irregular. Child showed marked prostration, in spite of the fact that the breathing was easier. It was thought best not to wait for the report of the culture, but give antitoxin at once; 1500 units were accordingly given. The further treatment consisted in cardiac stimulation, $\frac{1}{100}$ grains strychnia every four hours, tinct. strophanthus gtt i every three hours, calomel fumigations every three hours. Liquid nourishment. The prostration was so great that the writer spent a portion of the night at the bedside of the patient. On the following morning, second day under observation, the child was stronger, less dyspnœa, now chiefly inspiratory, temperature

101°, pulse 160, regular but feeble. Glandular swellings unchanged, spot of membrane on posterior pharyngeal wall disappeared. Bowels quite loose and green. Calomel fumigations discontinued. Treatment liquid nourishment, absolute rest in horizontal position. Strophanthus gtt ii every six hours. Strychnia sulphate $\frac{1}{10}$ grains every six hours. Cough still croupy, voice improving.

Third day under observation, child showed marked improvement, voice nearly normal, slight inspiratory dyspnœa; temperature normal.

Fourth day, patient considerably exhausted, otherwise well. Glandular swelling almost disappeared. No sequela. Culture showed diphtheria bacilli. Throat clear three weeks from onset. Urine negative throughout. No sequela.

The fifth patient was a boy three years old, strong and well-nourished, seen in consultation with Dr. W. B. Hoag. The child had a croupy cough and hoarseness for seven days. He was not sufficiently ill in the judgment of the parents to require the services of a physician. On the evening of the seventh day, he was taken suddenly with intense dyspnœa. Both inspiration and expiration were impeded. There was complete loss of voice. The writer was hurriedly summoned by the attending physician with a view to intubation. Upon my arrival the breathing was easier, but there was still considerable obstruction to both inspiration and expiration, but not enough to warrant intubation. There was but slight prostration, temperature 100° pulse 120, good. Very slight swellings at angle of jaw. The fauces were intensely congested. There was a small, thin membrane on the post-pharyngeal wall the size of a split plea. Culture taken.

TREATMENT.—Two thousand units of antitoxin were administered. Further treatment consisted of calomel fumigations, ten grains every three hours. Liquid nourishment.

On the following morning, the second day under observation, the dyspnœa was less marked, but still present. The patient seemed appreciably improved. The treatment with calomel fumigations was continued and 1500 units of antitoxin were given. Report of culture showed true diphtheria. Urine negative. Throat clear.

Third day under observation, temperature normal, patient quite bright, no prostration. As there was still a slight degree

of dyspnoea upon inspiration, 500 units of antitoxin were given. Throat clear, voice much improved.

Further history uneventful. Child well seven days after first injection. Throat free from bacilli. No complications or sequela.

The sixth patient was a boy two years old, well-nourished; seen in consultation with Dr. Geo. W. Jarman. First seen by the attending physician in the evening the first day of the disease. He had then a slight fever and a croupy cough. The next morning I saw the patient for the first time. The temperature was 101° to 102°, pulse 140, fairly strong, respiration 40. The voice was hoarse, the cough croupy; considerable dyspnoea; fauces congested; no membrane visible; chest clear.

TREATMENT.—Liquid nourishment; calomel fumigations, ten grains every three hours; strychnia $\frac{1}{160}$ grain every five hours. During the day the prostration increased. Respiration in the evening 60 to 70, pulse 150, temperature 102° to 103°. Stridor less, but voice hoarse and croupy. Antitoxin, 1500 units, given. Other treatment unchanged. Chest clear. At four o'clock the next morning, second day of illness, the dyspnoea became urgent. Intubation was performed. For an instant the rattling sound we like to hear, and then silence, and convulsive attempts at breathing on the part of the child, who became deeply cyanosed. The tube was hastily removed and found to be plugged with a tough piece of membrane the size of a dime. This was removed, and the tube re-inserted, with relief to the child so far as the obstruction was concerned. The respiration remained as rapid as before. In spite of the relief to the breathing, free stimulation and nourishment, the prostration increased. Rapid respiration was a prominent feature of the illness. During the day it ranged from 70 to 80 per minute. The chest had been repeatedly examined and showed no evidence of consolidation until eleven o'clock on the following morning. At this time we discovered a consolidation in the upper portion of the left lower lobe. From this time there was a rapid failure, the child dying from exhaustion at eight o'clock in the evening, forty-eight hours after the first sign of illness.

The writer feels that the lack of results in this case in no way militates against the value of antitoxin in diphtheria. The pneumonia and diphtheria evidently developed at about the same time, and was more than the patient could bear.

The seventh patient was a girl five years of age. Seen in consultation with Dr. Peter Murray. The writer was called by the attending physician to perform intubation second day of illness. Child was suffering from intense dyspnoea, both inspiration and expiration being impeded. There was marked prostration, pulse 150, irregular and soft. There was no visible membrane. Glands at the angle of the jaw slightly enlarged.

TREATMENT.—A tube was introduced with complete relief to the dyspnoea and 1500 units of antitoxin were given. Further treatment consisted of cardiac stimulation, with strychnine and whiskey. The tube was removed after an interval of six days without return of the dyspnoea. During this time the treatment consisted of liquid nourishment, strychnine and whiskey, with an occasional laxative. Culture taken the first day of the disease showed diphtheria bacilli. Two days later, the tenth day of the illness, the symptoms of slight fever and moderate dyspnoea with prostration reappeared; 1500 units were given. Intubation was not necessary. The active symptoms subsided, and the patient made an uninterrupted recovery, the throat being free from bacilli eighteen days after the onset. The temperature in this case was never above 102° F. No sequela.

New York Board of Health antitoxin was used in every case.

In conclusion, seven cases in all were treated, four boys and three girls. Four were intubated, with one death. Two others would have been intubated had I not felt it safe to use palative means, and wait for the effects of the antitoxin.

Initial dose of antitoxin was given: First day of disease, in one; second day of disease, in one; third day of disease, in two; fifth day of disease, in one; seventh day of disease, in one; after several days, in one.

The antitoxin was given the first day under observation in five. The second day under observation in two.

Two received single injection of 2000 units; two received single injections of 1500 units; one received two injections of 1000 units each; one received two injections of 1500 units each; one received three injections of 2000, 1000, 500, respectively.

RECOVERY CASES.—Signal improvement followed upon the use of antitoxin after an interval of twelve hours in four. After an interval of eight hours in another.

The remaining case showed no improvement in twenty-four

hours after the first 1000 units. This was repeated. Sixteen hours after the second injection, decided improvement was noticed.

113 WEST EIGHTY-THIRD STREET.

DISCUSSION.

DR. CAILLÉ.—Dr. Kerley's results are very good. The question that interests me is whether we need calomel fumigations when we use antitoxin. At the meeting two years ago I reported thirteen consecutive recoveries with antitoxin alone; a colleague in Brooklyn reported sixteen or eighteen cases, and a practitioner from Newark reported twenty-three consecutive recoveries with antitoxin alone. If the calomel fumigations plus antitoxin are better than the antitoxin alone, we ought to adopt them. If they are not any better, I would say we ought to drop them.

DR. FRUITNIGHT.—In answer to Dr. Caillé's remarks, I would say that I was an ardent advocate of calomel fumigation in the treatment of laryngeal stenosis before the introduction of antitoxin, but since then I have not used fumigation, but have relied altogether upon antitoxin. It is just in these cases I think that antitoxin shows its brilliant results. For in these very cases in which laryngeal stenosis is threatened, it has been my experience that the use of the diphtheric antitoxin has prevented the full development of diphtheritic croup, frequently rendering intubation unnecessary, whereas formerly nearly all pronounced cases of this description would be obliged to be either tracheotomized or intubated.

THE ENANTHEM OF GERMAN MEASLES.

BY F. FORCHHEIMER, M.D.,

Professor of the Practice of Medicine and of Pediatrics,
Medical College of Ohio,
Cincinnati.

There is a certain number of excellent observers who still express doubt in regard to the nosological position of rubella; Hensch, the Vienna school of dermatologists (Hebra and Kaposi), even some of our own members (Townshend and others, personal communications) look upon rubella as either a diminutive form of scarlatina or of measles, or as a resultant manifestation of a combination of both poisons.

To those who accept rubella as an independent morbid entity, the evidences have been sufficiently weighty to permit them to formulate their conclusions; additional evidence, however, will not be unwelcome, and not the least important evidence would be the detection of a characteristic enanthem in this disease. The study of the enantheims in the acute exanthematous diseases, has assumed formidable proportions within the last ten years.

In the observations of skin manifestations, twenty-five years ago, we were all so dominated by the overwhelming, masterful descriptions of Hebra and his followers, and by the theorem that, in the acute exanthemata, the same lesion is always produced by the same poison, that the manifestations upon mucous membranes were treated in a decidedly off-handed way.

It was only late in the seventies that the Vienna school of children's physicians began, and, it may be added, completed their fight against the dermatologists in connection with the establishment of varicella as an independent morbid entity, and from this time on, we have begun to look upon the eruptive fevers in a broader way.

As a result, the other symptoms of the acute enanthemata have received more attention, so that at the present time, the enantheims can be utilized for differential diagnostic purposes, and,

as a corollary, decided help to the location of these individual diseases in the nosological table is added.

This is true for measles, scarlatina, small-pox and varicella. For rubella, as will be seen, much confusion exists, and while various observers have seen the enanthem, I have failed to find a description of its peculiarities and its life course in literature. Indeed in the vast majority of descriptions it is supposed that rubella is characterized by the absence of an enanthem. One of the first authors who has studied the question (*Schwartz, Über ein charakteristisches. Differential merkmalzwischen Morbilli und Roseola, Wiener Med. Presse, 1868*) finds isolated reddish spots on the hard and soft palate and uvula in measles, but not in rubella. Thomas (*Beobachtungen über Rötbeln, Jahrbuch für Kinderheilkunde, ii., N. F., 1869*) promptly disavows this, and makes the statement that there is no specificity of the enanthem in either disease; that, therefore, it cannot be utilized for purposes of differentiation, and that any difference between the two is simply that of intensity, in that it is better developed in measles than in rubella.

In a subsequent communication, Thomas (*Neue Erfahrungen über Rötbeln Jahrbuch für Kinderheilkunde, N. F., v., 1872, page 346*) again comes back to the question of the enanthem and the appearance upon the mucous membranes in rubella, and says "injection of the mucous membrane of the palate was never absent, the pharynx was injected, as a rule; sometimes the tonsils were slightly swollen and then there were slight and transitory disturbances of deglutition.

"The injection, now as before, did not seem to be macular. I must, therefore, according to my recent experience, designate the term enanthem (*Schleimhaut exanthem*) as unsuitable, in so far as conveying the meaning of a something like the exanthem. Moreover the affection of the mucous membrane is streaked and spotted, individual injected portions being separated by others less injected or normal.

"The appearances upon the mucous membrane do not materially differ from those in measles and do not, then, admit of differentiation, as has been claimed. The injection begins with the first development of the eruption and disappears rapidly when it fades."

Emminghaus (*Ueber Rubellen Jahrbuch für Kinderheilkunde, N. F., iv., 1871*) has evidently seen an enanthem, in that

he says: "In a few cases there was observed upon the palate a similar exanthem (*i.e.*, similar to the one upon the skin), which was always circumscribed and strictly limited." Dunlap (*Lancet*, ii., page 464, 1871) makes a most concise and explicit statement, as follows: "I then found that in the great majority of cases the velum, the uvula, and the fauces generally, were covered with a deep red, punctate eruption."

Nymaan (*Zur Aufklärung de Frage über die Selbständigkeit der Rubellen*, *Oest. Jahrbuch für Kinderheilkunde*, iv., page 125, 1873) describes a condition of small punctated redness upon the pharynx, the hard and soft palate as the enanthem; a condition that is found in various exanthemata, in ordinary catarrhal stomatitis, even in apparently healthy mouths. As Nymaan has been quoted quite frequently, it may be well to state that he, at least, has not seen an enanthem.

Gerhart (*Lehrbuch der Kinderkrankheiten*, 1881, page 82) describes the enanthem as follows: "The mucosa of the pharynx presents the same spotted-hyperæmic, hemorrhagic punctated redness as in measles," a condition which, according to the view to be presented, is just the one that does not exist in rubella.

Kassowitz (*Tran. International Congress*, iv., page 13, 1881) is also one of the few who has seen the enanthem, in that he says that "in a small number of cases he has observed isolated red spots upon the mucous membrane of the hard palate during the first day of the disease." J. Lewis Smith, at the same meeting, stated that "catarrhal hyperæmia is also seen in spots or patches, more or less diffused upon the buccal surface."

Rehn (*Eine Rubeolen Epidemie*, *Jahrbuch für Kinderheilkunde*, xxix., N. F., page 282, 1889) has also seen something which he describes, occurring in nearly all cases as "the granular swelling of the glandular and speckled form."

Picot (*Jaccond's Dict. de Médeciné*, xxxi., page 774) describes the enanthem as follows: "It may appear upon the velum, the palate, similar to that of measles; we have seen it once, but have been able to verify its absence in other cases."

We now come to those excellent and exhaustive papers on the subject of rubella, written by J. P. Crozer Griffith (*The Medical Record*, 1887), J. E. Atkinson (*American Journal of Med. Sciences*, 1887), and W. A. Edwards (*Keating's Cyclopædia of the Diseases of Children*, Vol. i., page 695). Griffith has seen

one phase of the enanthem in that he mentions, in a few instances, "an eruption of small, yellowish-red or brownish-red spots of pin-head size, was visible over the soft palate and uvula and the inner surface of the cheeks."

Atkinson, in discussing Schwartz (referred to before), says: "This undoubtedly holds for measles, but it is valueless for diagnosis, for the throat eruption of r \ddot{o} theln often exactly resembles that of measles. It is more often diffuse than macular." Edwards makes the statement that he has "also found in some cases an eruption scattered over the throat; this has been noted by several other observers, and we may note an eruption in the throat preceding the cutaneous eruption." Barthez and Sanné (*Traite clinique et pratique des Maladies des Enfants*, iii., page 68, 1891) have noted a little palative redness and some laryngeal phenomena "seeming to indicate an enanthem." Filatowtil (*Vorlesungen über Acute Infections, Krankheiten im Kindesalter* translated from the Russian by Polonsky, viii.-ix, page 424, 1896) has no doubt "that the eruption is met with upon the soft palate, indeed, upon all the visible mucous membranes; here also occasionally in the form of punctated hemorrhages, although many authors deny this."

After the review of the literature, it will be seen that no conclusion can be drawn as to an enanthem in rubella, and the whole question is dismissed in a most characteristic way by Jürgensen (*Northnagel's specielle Pathologie u. Therapie* Vol. iv., iii., ii., page 97, 1896) in the following sentence: "An enanthem may be present—it may also be absent," to which might be added this: an enanthem may be characteristic—it may also be not characteristic.

On account of the fact that the majority of observers either does not mention an enanthem, or confuses it with that of measles, and, furthermore, because of the confusion that exists in the description as it is given in those authors mentioning an enanthem, I decided to study this question upon the first available opportunity. This occurred last winter in a wide-spread epidemic of rubella, which occurred in Cincinnati.

It is unnecessary for my present purpose to describe this epidemic, except in so far as to state that it was characterized by the presence of all those symptoms that can be considered classic by the authors; that it was accompanied, in my own experience,

by no mortality; that in only one case did I see a complication (nephritis), and that I should call it rubella morbillosa. In order to afford me further evidence that the epidemic was one of rubella, we are now (May, 1898) having an epidemic of genuine measles, and up to the present time, I have seen seven children attacked by the latter, who, two or three months before, have had rubella.

In the first case of rubella which I examined, I was completely foiled as to the detection of an enanthem and could discover nothing more than has been described by the authors which, for the most part, I have always considered as neither conclusive nor distinctive.

It was not until one of my own children was attacked by the disease, that I was enabled to see an enanthem, to study it and, at the same time, to offer an explanation for the fact that it had not been described more accurately before. After this there was no difficulty in detecting this enanthem in all cases, provided the conditions were favorable. The first child, a boy of eleven years of age, went to bed feeling perfectly well; in the morning he took very little breakfast, refused to play, preferring to lie upon the sofa, had a slight elevation of temperature, complained of a little of sore throat and said he was dizzy. Upon an examination, the tonsils were found slightly reddened, and the glands at the angle of the jaw decidedly enlarged.

As the boy is subject to attacks of angina follicularis, it was supposed that he was coming down with this trouble, and, in the course of my morning rounds, two hours after the first inspection, I stepped in to see him again. I then noticed that he had a rash upon the lower part of his face which, to me, was characteristic of rubella. Upon examining his mouth, I found a macular, distinctly rose-red eruption upon the velum of the palate, the uvula, extending to but not on to the hard palate. These spots were arranged irregularly, not crescentically; were the size of large pin-heads, were very little elevated above the level of the mucous membrane, and did not seem to produce any reaction upon it.

Six hours after this, the enanthem had become decidedly paler, the infiltration very much less, and by the next morning it had disappeared.

The exanthem by this time had developed in its natural way, and the case ran its course as an ordinary one of German

measles. After having learned the lesson that the enanthem appears simultaneously with the exanthem, I took occasion to study it in subsequent cases—twenty-two in all, in which the time factor was favorable.

It will be necessary first to dispose of the buccal appearances in rubella. They are seen in the mouth as catarrhal stomatitis, streaking and spotted appearances mentioned by authors referred to before; upon the pillars of the fauces, both anterior and posterior, in the form of injection, or more decided hyperæmia; upon the tonsils, from an ordinary catarrhal angina, with swelling to a streptococcus amygdalitis (there is no reason why true Loeffler diphtheria should not develop, but I have not seen it); upon the posterior wall of the pharynx, in the form of acute catarrhal swelling, with hypersecretion, or any other process that may be going on in the mouth at the same time.

As the result of my previous, as well as my present, investigations, I do not hesitate to pronounce all these manifestations as due to secondary infection and as absolutely irrelevant as to the poison of rubella, except in that the latter acts as a predisposing factor for the development of other morbid agents.

I do this the more positively as I have seen the whole specific process run its course upon exceptionally pale mucous membranes without there being any secondary reaction—indeed, it is in these that the enanthem can be seen to its best advantage.

The results of my studies, which I beg to present to you, are as follows: Because there is practically no period of invasion in rubella, the enanthem appears at the same time as the exanthem. In no case have I seen the enanthem when there was not present a suggestion of the enanthem. It may be that further study in other epidemics may lead to different observations in this as in all of the other points I shall make, for undoubtedly epidemics of rubella differ very much, possibly more than any of the other acute exanthematous diseases, in their manifestations.

The enanthem is very short-lived; it fades away within the first twenty-four hours, and then come certain results of involution, not present in the majority of the cases. It is localized upon the uvula and soft palate and rarely invades the hard palate; I have never seen it extend to any other part of the mouth.

It is the same eruption that is found upon the skin, characterized then by its size of efflorescence, its arrangement, the

absence of great infiltration and, above all, by its color, this being a pure pinky rose-red, almost exactly the same as the roseola of typhoid fever.

During the process of involution there are sometimes left pigmented deposits, usually of a yellowish, or yellowish-brown color, either in the form of spots or streaks.

The claim that this enanthem is distinctive can be defended by comparison with the enanthem of those two diseases with which rubella is confounded. A glance at these will suffice to establish this proposition. In scarlatina the enanthem appears from twelve to twenty-four hours before the eruption; it appears on the pillars of the fauces in the form of the characteristic puncta, then rapidly spreads over the mouth in the form of a scarlet red coalescing eruption, which finally ends in desquamation, producing the strawberry tongue, and lasting well into the second week of the disease.

In measles the enanthem begins upon the soft palate from thirty-six to forty-eight hours before the exanthem, in the form of purplish or bluish papules, arranged crescentically, extends over the cheeks, accompanied by the blue tongue; it is at its maximum with the beginning of the eruption and may take as long as three or four days to disappear.

It will be seen, therefore, that in all respects does the enanthem of rubella differ from that of scarlatina and measles, and when seen can be utilized with certainty for differential diagnostic purposes.

It is not within the scope of this paper to discuss the fact whether an enanthem can or cannot be observed in all cases of the acute enanthemata, and, in conclusion, I wish again to call attention to the statement made before, that these studies were made in one epidemic only and their verification must rest with the study of other epidemics before they can be accepted as belonging to all cases of rubella, under all circumstances.

DISCUSSION.

DR. J. P. CROZER GRIFFITH.—Dr. Forchheimer has mentioned two facts which help to explain something of the differences in the descriptions of writers who have referred to the occurrence of this enanthem. As he says, truly the majority of writers do not mention it at all. I have heard physicians make the statement that a differential diagnosis between rubeola and rubella

may be made by the fact that rubella does not have any eruption in the throat. This of course we know not to be true. One of the two facts mentioned by Dr. Forchheimer, to which I would refer, is the multiform character of the cutaneous eruption. Certainly there is no one of the exanthemata of which this characteristic is truer. It is very likely the case that this applies also to the eruption in the throat. One author may describe it in one way and others in other ways, and all be right. The other fact to which he refers, and which I would mention, is the fleeting character of the rash on the skin. There is no disease I suppose which has a rash of a more transitory nature than rubella, at least in many instances. I have often been struck by the wave-like manner in which it spreads over the body, disappearing in one place before it has fully appeared in another. One may, it is true, often find patients with a rash which seems to be well developed all over at one time, and which is quite persistent; but certainly in very many cases it spreads, as I have said, like a wave. In the presence, then, of this fact we can readily expect to see the eruption in the throat lasting perhaps for only a very short time. That may account for the fact that it is frequently overlooked. I believe it is often present, but not found. I do not know whether I would go quite as far as Dr. Forchheimer in the belief that the diagnosis may be made so frequently by the character of the eruption in the throat, because I have repeatedly seen an eruption on the skin in rubella which I could not tell from that of measles, and it is very probable, therefore, that the eruption in the throat may also resemble it exactly sometimes. But I think he is quite right in describing the average eruption as a pale and fleeting rash on the palate in rubella, and as a much more distinct, larger and deeper red eruption in measles.

DR. W. P. NORTHRUP.—I would like to know the definition of enanthem.

DR. FORCHHEIMER.—It is a term that has been introduced especially by the French authors, and justly introduced I believe, simply meaning an eruption on the membrane, when it is an enanthem, and when it is without, it is an exanthem. That is the only difference between the two. I have used the term constantly and I think we all have, and doubtless Dr. Northrup has himself.

DR. NORTHRUP.—No, I have not.

DR. FORCHHEIMER.—Then you soon will.

DR. A. JACOBI.—Dr. Northrup makes a similar difference himself, for instance, between epithelia and endothelia, for the same reason that we do between exanthem and enanthem.

DR. E. M. BUCKINGHAM.—In a very large number of cases of measles that I have watched from day to day, I have found that

on drawing a line with pen and ink around the individual spots, it proved very conclusively that the same spot is not always the same size. This makes a very distinct division between the two.

DR. SAMUEL S. ADAMS.—My experience coincides with that of Dr. Forchheimer so far as the spots are concerned, and particularly in reference to the rapidity with which they disappear. Of course the best way to study the enanthem is in the negro. I have observed that these spots were present on the mucous membrane; in German measles it disappears very rapidly and in measles it follows the true course of the disease. Another point is the character of the eruption upon the throat as compared with that on the external surface. I think we will find by observing the cases closely, that it is, to a very great extent, an index. If the eruption upon the throat is severe, then the eruption upon the skin will be severe. That possibly will be found true of measles, but I have not observed it as much. Certainly it has been present in epidemics. In one epidemic in the Foundling Hospital we had fifty children affected with it without any mortality whatever. That occurred about two years ago and was followed by an epidemic of measles, which my predecessor attended, in which the mortality was very great.

DR. T. M. ROTCH.—If this symptom, or rather sign, of Dr. Forchheimer proves to hold good in coming epidemics, of course it will be a very valuable addition to our means of diagnosticating the disease. There are a great many points in the discussion which usually take place when we speak of r \ddot{o} theln and measles, on the side of those who hold that there are diagnostic points. Most observers state that they have an epidemic of rubella and then proceed to describe it. In many instances it seems that they had no right to call it r \ddot{o} theln. Many observers fail to recognize that in measles there is a great variety in the symptoms. Those who attribute diagnostic value to some of these symptoms of r \ddot{o} theln should also at least allow that measles is very varying, and that these same symptoms are found also in measles. The symptoms also vary much in intensity in measles. If the symptom referred to by Dr. Forchheimer is proved to be diagnostic, a decided advance will have been made in recognizing the disease. Undoubtedly measles may occur a number of times.

DR. A. JACOBI.—I cannot speak from my own experience in these matters, but it strikes me that this paper of Dr. Forchheimer is a very valuable addition to our means of diagnosis. We all know how difficult it is to make up our minds oftentimes in cases resembling measles. There are two additions to our means of diagnosis that have come up the last year: First, the diagnostic enanthem referred to by Dr. Forchheimer, and second, the enanthem described by Koplik in measles. In re-

gard to that I will say that again I have no experience, but I have talked with a number of men who have seen the same enanthem in the mouth, about the cheeks, that Koplik describes. Moreover, there is confirmation of this on the other side of the water by very good observers. In the Berlin Children's Clinic, under Heubner, every case of measles has been examined for these signs. The report, by Slawyk, may be found in one of the last numbers of the *Deutsche Medicinische Wochenschrift*. Of twenty-six cases of measles they found it, if I am not mistaken, constantly in twenty-five cases, which would make 96 per cent. If Koplik's enanthem appears to be confirmed for measles, and there is another enanthem which can be recognized as characteristic of rubella, then I think the mucous membrane will be more safe to use for the purpose of diagnosis than the external integument.

DR. CAILLÉ.—Dr. Forchheimer's suggestions may be of value to us in making an early diagnosis, but the term enanthem is one I do not like to see made use of generally. We have no visible rash where there is endothelium. We have visible rash where there is skin and mucous membrane. In regard to the possibility of telling one form from the other early, I have been trying that very earnestly for the last ten years, as it is one of my duties to keep measles, scarlet fever, rubella, and all forms of contagious disease out of the "Babies' Wards," and I have come to the conclusion that we will always be able to distinguish one form of rash from the other in the majority of cases, but in some *we will not be able until we have a culture test.*

DR. HENRY FRUITNIGHT.—Since I have read Dr. Griffith's contributions, I have examined the mouths of all the cases of rōtheln in German measles which have come under my care, and found the signs described by Dr. Forchheimer present in more than 70 per cent. of the cases, and I am inclined to think it is a diagnostic test. As a proof of this I will say that in some cases the patients had passed through attacks of true measles several weeks before the attacks of rubella, and the appearance of the enanthem in the mouth was entirely different from that present during the attack of rubella. Furthermore, we have had quite an epidemic of measles in the section of New York in which I reside and in 90 per cent. of the cases the eruptive symptoms in the mucous membrane of the mouth, as described by Koplik, have been found. In regard to the symptoms differentiating between rubella and rubeola, I would say that the symptoms on the mucous membrane of the mouth are much more aggravated in measles. Very often in rubella there is almost a total absence of constitutional symptoms, and this, together with the rapidity with which the rubella eruption disappears, is another diagnostic point.

DR. FORCHHEIMER. —I have not referred to Koplik's enanthem, because I want to verify it myself. I do not doubt he is right. I have in my little book on the Diseases of the Mouth, some time ago, described the appearances in the mouth. It seems to me if one has once seen these and taken into consideration the appearance of the lips, cheeks and tongue, it is almost impossible to mistake these manifestations for those of German measles. The two conditions are to me so absolutely characteristic and distinct that I am always prepared when I see the condition in German measles or genuine measles, to be willing to make a diagnosis before the eruption appears; in genuine measles, sometimes as long as thirty-six hours. In regard to the objection of Dr. Buckingham, of course I am describing the average when I say they are the size of a large pin head. I have seen them smaller, but I have never seen them larger. Above all is a peculiar color. The color of the enanthem is different from that of measles, and altogether different from that of scarlatina. Any one who has seen it cannot fail to distinguish between these enanthemata. In the enanthem of German measles the spots do not increase in size. When they come out they come out in their largest circumference, and then there takes place a process of involution, which, as I stated, sometimes in very pale mouths leads to pigmentation, just as pigmentation may take place in the skin.

DERMOID CYST OVER THE CENTRE OF THE LARGE FONTANELLE.

BY A. JACOBI, M.D., LL.D.,

New York.

Av., a Cuban boy, was presented when he was eight months old, with a tumor of the size of a coffee bean over the centre of his large fontanelle. It was first noticed when the child was a few months old; at that time it had the size of a pea. The fontanelle was open and of the size it usually has at the age of eight months; pulsation could be felt through it, and crying raised it. The tumor was of the color of the scalp, not vascular, not congested, not sensitive, and covered with hair. It was not changed in size or shape by compression, but could be depressed (the fontanelle being still fibrous). Some pulsation could be felt through the tumor, and the latter would rise with forced expiration (crying). These symptoms were attributed to the condition of the fontanelle on which the tumor was situated, but the operation was postponed on account first of the possibility of a mistake, and of the increased safety of a surgical procedure in later years when ossification would have been completed.

The child was again presented in May, 1898, when he was four years and nine months old. The tumor had the size of a hazelnut, was covered with hair, not discolored, not vascular, not markedly depressible, but elastic and semi-fluctuating, and slightly compressible under bi-lateral pressure. It was not transparent. Its shape was spherical; it had no pedicle, but its base appeared narrower than the rest of the tumor. It was not removable from its point of attachment, which was quite firm. The skin on top, at the greatest distance from the skull, appeared rather thin. There was no pulsation in or through the tumor, and no change with exertion or with crying.

DIAGNOSIS.—Congenital dermoid cyst. No possible mistake for meningocele.

A longitudinal incision through the covering scalp proved the skin of normal thickness. The capsule was easily found

and readily separated from the surrounding tissue until the periosteum was reached. There it and the capsule were firmly adhering. In the attempt at separating them a small opening was accidentally made into the tumor, and some little of the contents was lost. The opening was kept closed with pincers. The periosteum was torn off the bone to the extent of a square cubic centimetre. This part of the periosteum which remained attached to the tumor was rather thin. During the latter part of the operation the cause of the difficulty in finishing it quickly was found in the fact that there was a depression of the bone more than half a centimetre in depth in which the lower part of the tumor was imbedded. The bone itself, with the exception of this depression, was normal; there was no hyperostosis around the depression, or any where else.

The tumor was found to be a cyst, both dermoid and sebaceous. Inside the part bordering on and attached to the indentation of the bone, there is a small bundle of minute hair. The microscope shows fat globules in large quantities and cholestearine crystals.

The locality of the tumor, and its contents, prove its dermoid character. It having been observed a very few months after birth, when it was quite small, and the depression of the bone, prove that it existed at an early time. There can hardly be a doubt that the duplicature of the ectoderm forming the cyst was of early foetal nature. It is probable that where the tumor was formed ossification remained incomplete and the bone thin. In a case of Heurtaux's, in which the development of the tumor became rapid about the thirtieth year of the patient, there was no ossification at all. In most other respects the description of my case is identical, to a great extent, with all those reported. In a case of Giraldés, and in one of Arnott, there was the same apparent pulsation which I described above. In a few cases the bone was found very thin, either by absorption or by deficient ossification; in a few others there was a hyperostosis round the point of attachment in the periosteum.

The diagnosis from encephalocele, or from meningocele, may be doubtful in occasional cases. The scarcity of the former over the centre of the fontanelle should not be claimed as a diagnostic point, for cases like that described above are also rare. A meningocele fluctuates, is very transparent (but possibly may, when small, be covered by skin), hairless, and compressible;

its contents can be forced back into the cranium, and may thus give rise to cerebral symptoms. Traumatic "spurious" meningocoele need not give rise to cerebral symptoms, may not be (rarely is) reducible, and is covered by normal skin. When very small, it may give rise to doubts, and in some cases, when the question of an operation is raised, it will not do to be rash.

The contents are not always of the same consistency. Some contain, with the usual contents, a serum rich in sodium chloride.

A case of Sibthorpe and Hardy was not exactly over the centre of the fontanelle.

The number of cases of dermoid of the large fontanelle which have been reported is small. The first case, by Heldea (1770), was quoted by Wernher, who took these tumors to be strangled meningocoeles. Picard related one in 1840; it was as large as a fist, and was observed on a woman of sixty years. Hewitt collected five cases, Giraldês fourteen. The best article on the subject known to me is that by Lannelongue and Ménard, in their "malformations de la tête et du cou."

HEATSTROKE IN INFANTS.

BY IRVING M. SNOW, M.D.,

Clinical Professor of Diseases of Children, Medical Department, University of Buffalo ; Member of American Pediatric Society ; Physician to Buffalo Fresh-Air Mission Hospital, etc., Buffalo, N. Y.

It is unfortunate that the condition known as heatstroke is so rarely described in the numerous treatises upon diseases of children. Most books upon general medicine discuss the subject only in reference to adults and adolescents, and the reader would infer that the symptoms of heatstroke were never observed in infants or young people.

A few illusions to insolation-heatstroke in babies may be found in periodical literature.

Illoway records three cases of heatstroke in infants. Dr. Henri La Fleur presented the history of a case of insolation before this society in 1896. The discussion of the paper developed the fact that in Washington, New York, and Philadelphia, heatstroke was occasionally observed in babies. The interesting point was also raised, whether the hyperpyrexia and cardiac depression observed in Dr. La Fleur's case were not due to a gastro-intestinal toxæmia.

The two conditions are easily confused, and the border-line between the two maladies is exceedingly vague, as frequently in infants and occasionally in adults the hyperpyrexia of heatstroke is associated with vomiting and diarrhœa. It is well to remember that in dog-days, some so-called cases of cholera infantum with excessively high temperature and moderate vomiting and diarrhœa are actually cases of insolation, and that prompt recognition and vigorous hydrotherapy are necessary to save life.

During July, 1897, I observed two cases of heatstroke in babies. For the first few days in July the weather was excessively hot, the average maximum temperature from the 3d to the 10th of July being 90.4° ; the air was still; the sky clear; the atmosphere not unusually humid. As a result of this heat twenty-four deaths from heatstroke occurred in Buffalo—an unprecedented number for Buffalo, where the malady is rare.

METEOROLOGICAL OBSERVATIONS, TAKEN DURING THE MONTH OF JULY, 1897.—BUFFALO.

Date, 1897.	BAROMETER.		THERMOMETER.					RELATIVE HUMIDITY.		WIND.				Rain or melted Snow, inches.	STATE OF WEATHER.	
	8.00 A.M.	8.00 P.M.	8.00 A.M.	8.00 P.M.	Max.	Min.	Mean.	8.00 A.M.	8.00 P.M.	Direction and velocity in miles, per hour at		Daily Velocity Movement	8 A.M.		8 P.M.	
										Dir.	Vel.					8.00 A.M.
1	29.06	28.97	66	74	80	64	72	94	60	S	2	W	3	118	Pt. Cl'dy.	Clear.
2	28.98	29.02	69	74	82	67	74	78	84	SW	8	SW	8	175	Sprink'g.	Clear.
3	29.16	29.15	75	88	93	70	82	72	43	S	8	SE	6	164	Clear.	Clear.
4	29.22	29.16	81	87	91	78	84	61	60	S	18	S	12	305	Clear.	Clear.
5	29.20	29.12	81	85	87	74	80	72	50	S	9	SW	9	333	Clear.	Pt. Cl'dy.
6	29.29	29.29	76	83	86	72	79	61	62	N	5	NE	5	220	Pt. Cl'dy.	Clear.
7	29.35	29.28	75	84	92	68	80	58	38	E	8	NE	12	218	Cloudy.	Clear.
8	29.28	29.17	79	86	92	69	80	60	42	SW	7	NE	7	165	Clear.	Cloudy.
9	29.17	29.09	78	81	87	76	82	67	76	SW	8	SW	6	196	Pt. Cl'dy.	Pt. Cl'dy.
10	29.11	28.99	82	88	95	76	86	58	55	S	6	SE	7	145	Clear.	Pt. Cl'dy.
11	29.81	28.86	79	67	83	64	74	62	97	S	20	NW	14	379	Cloudy.	Cloudy.

CASE I.—A boy of six months; weight fifteen pounds. In early infancy he was suckled for a few weeks and afterwards fed upon diluted cow's milk. At the age of nine weeks he suffered from a paroxysmal cough (pertussis?). During the first three days in July the baby showed signs of indigestion—vomiting, and passing dyspeptic stools. July 4th, the atmospheric temperature was 93° , the hottest afternoon of the season; the child was taken three miles in an electric car. While on the car he looked very ill and had a convulsion. I saw him at 5 P.M.; there was a rectal temperature of 107.6° ; respiration 60; the heart's action was rapid, feeble, irregular; there were moist râles at the base of the lungs; the skin was hot and burning; the complexion became gray; the child was in most desperate collapse.

The baby was placed in a bath at 80° F., five minutes' friction being used; it was then placed in a wet pack for twenty minutes; after the pack the temperature was 105° , one hour later 99° . The baby was given thirty minims of whiskey hypodermically; the feet were wrapped in hot flannels. Nevertheless, there was no improvement in the child's general condition.

At 7 P.M., temperature, 103° ; respiration, 40; cold-pack and intestinal irrigation at 80° ; at 9:30 P.M., temperature 105° ; bath and pack twenty minutes later, temperature sank to 99° .

During the night the child was kept out of doors and continually fanned. Temperature taken hourly, varying between 101° and 102° ; the baby was given whiskey and water frequently, and also a laxative dose of calomel. July 5th, temperature 100° , 101° , the child's expression was better, the pulse stronger. It had two blackish stools during the day; was fed upon whiskey and water and chicken soup.

July 6th.—Temperature 100.5° ; occasional cough; râles at the base of the lungs; no rapidity of respiration or areas of silence; two dark watery passages. The trunk was covered with a punctate, scarlet rash. Food taken with avidity.

July 7th.—All symptoms improved.

Rapid and complete convalescence; no peeling of the skin. This child presented only the symptoms of high temperature and profound collapse; its condition was quickly improved by hydrotherapy; the bronchitis was a relic of an antecedent whooping cough; the scarlet rash an erythema due to the general disturb-

ance of function from the hyperpyrexia, although the baby was under weight, badly fed, and dyspeptic; there were only mild digestive symptoms.

CASE II.—Boy; seven months old; suckled; well, with exception of colic.

The child slept in a room directly under a flat tarred roof. During the hot days in July the tar melted and flowed down the sides of the building. In this sweat-box the baby lay during the hot weather.

Its illness was first noticed July 5th, when the child ceased to perspire, refused its food, and a rash of widely separated papules appeared upon the back of the neck, and later spread over the entire trunk. The baby seemed somnolent during the morning, but started whenever an explosion of cannon or fire-crackers occurred near its room. Its skin was hot and burning to the touch; the bowels were constipated.

July 6th.—A physician saw the child; found a rectal temperature of 107° and sent the baby to the General Hospital, service of Dr. Himmelsbach, by whose kindness I am enabled to report the case.

At 1:30 P. M., received at hospital; child plump, well developed, crying, restless; skin hot and dry; pupils dilated; occasional muscular twitching; temperature 106.2° ; pulse 176; respiration 78° ; eruption very noticeable.

The baby was given a cool intestinal irrigation; was placed in a cool bath at about 60° ; was well rubbed; afterward it was taken out of the bath and given fifteen minutes' friction with a dry towel. Before the bath, and one and one-hours afterward, it received $\frac{1}{80}$ grain strychnia, hypodermically.

The patient reacted well; one and one-half hours after the bath, temperature 99.4° ; it was given whiskey, chicken broth, ice was placed on its head. At 5 P. M. the child was still unconscious; fair pulse; no fever.

Temperature at 8 P. M., 104.4° ; at 11 P. M., 105.2° ; the hydro-therapy was continued. Potassium bromide 0.06 given at 11:30 P. M.; at 8 P. M. with the rising temperature the child began to have loose, watery passages, which occurred every half hour, until its death at 1:30 A. M.; autopsy refused.

This child, breast fed, of superb physique, after lying three days in a close, hot atmosphere, developed somnolence and high

temperature. No improvement was effected by the reduction of the temperature by hydrotherapy; the temperature quickly rose and was associated with profuse watery stools. The diarrhœa was probably a terminal symptom from acute impairment of the digestive functions. Occurring in a breast-fed child already moribund, the diarrhœal condition bears but slight resemblance to cholera-infantum.

The writer, while clinical clerk in the Great Ormond Hospital for sick children, saw a case similar to the first described in this paper. A poorly nourished baby was brought to the hospital in a convulsion; temperature 108° . Cold baths were employed; the child, nevertheless, showed a recurring tendency to hyperpyrexia for eighteen hours; the fever finally subsided; there was no disturbance of digestion, nor did any acute malady subsequently develop; the patient was discharged, cured. From its actions and its micro-cephalic head the baby was supposed to be a congenital idiot. The illness bore an exact resemblance to the hyperpyrexia of heatstroke, although it occurred amidst the dense fogs of a London autumn.

It may be again emphasized that heatstroke in babies is easily confused with an acute gastro-enteric infection. The temperature is often not accurately taken or observed at all. The rapid collapse is ascribed to the very moderate vomiting and diarrhœa.

In typical heatstroke if the patient be thoroughly examined, the whole attention of the physician will be centered in the extraordinary high temperature; the loose passages occur late, and are terminal symptoms; the cardiac depression and temperature, 107° to 108° , are not sufficiently accounted for by the usually moderate diarrhœa; again, in an intestinal toxæmia, the fever commonly subsides after vomiting and purging.

The therapeutics of insolation are well understood. In babies in addition to baths and packs, intestinal irrigations with cool water 70° to 80° , are to be recommended. It is also probable that the subcutaneous injections of large quantities of artificial serum would revive the flagging strength of the heart, or would combat the unknown infection or toxæmia which, Sambon has recently asserted, is the cause of the group of symptoms which he calls, *siriasis* (heatstroke). The fact that injections into the cellular tissue of large quantities of artificial serum, have great curative powers in mycotic vomiting and diarrhœa, and in ileocolitis has been recently clearly proved by Le Sage. In infantile

heatstroke this procedure would be indicated, if no improvement had occurred in the cardiac respiratory and nervous symptoms, after the temperature had been reduced by hydrotherapy.

Dr. L. Westensa Sambon, of London, advances the theory that heatstroke is an infectious disease. Whether it be an auto-infection, some micro-organism under the influence of high atmospheric temperature acquiring extreme virulence, or whether the avenue of infection be the respiratory or digestive tract, Dr. Sambon does not show. He believes that heatstroke should be placed in the same category as yellow fever and dengue, being due to a specific germ, to whose growth and transmission to man a high atmospheric temperature is necessary.

Dr. Sambon asserts, that many facts point to the infectious nature of heatstroke, viz.: the relative immunity of residents in subtropical and tropical climates, the epidemic prevalence of heatstroke, not always at the hottest time of year; also sundry nearly uniform pathological findings, the fluidity of the blood, the peculiar engorgement of the lungs, the ecchymosis in various organs, the acute parenchymatous degeneration in nervous and other tissues, are highly characteristic of acute microbial-poisoning.

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476 FRANKLIN STREET.

FATAL HÆMOPHTYSIS IN AN INFANT TWO YEARS OLD.

BY GEO. N. ACKER, A.M., M.D.,

A. H., aged eighteen months, male, colored, came under my care at the Children's Hospital, December 21, 1896. The infant was treated in the hospital during the summer for enteritis and was discharged cured October 23d. He remained well until the middle of September, when a cough developed, which has been getting worse up to the present time. The patient has been losing flesh and is emaciated and anæmia. He has a frequent loose cough. There is slight dulness with some mucous râles at the base of the right lung anteriorly. There is dulness over the left lung anteriorly with few râles. The respirations are 64 per minute. The pulse is 160 per minute, and the temperature 104° F. The tongue is slightly coated. For some days he has had three loose stools daily. The infant has been given general diet.

January 18th.—The patient has failed to improve. There is dulness over the left lung anteriorly, and posteriorly, with diminished breathing and many mucous râles.

There is slight dulness lower part of right lung with bronchovesicular breathing, and numerous fine mucous râles.

There has been an irregular temperature ranging from 99° F. to 103° F. On several dates the morning temperature has been 97° F. and the evening temperature 105.5 F.

The condition of the infant gradually became worse, and the pulmonary symptoms more marked, when he suddenly died May 6, 1897, from a profuse arterial hæmorrhage from the lungs.

Necropsy made by Dr. Walter Reed, U. S. A.

Examination shows numerous areas of tuberculous caseation, split pea in size and larger, scattered through substance of both lungs. Upon opening trachea there was found on posterior wall an ulceration, oval in shape, its long axis parallel with that of trachea size 13 x 6 m.m. extending through mucosa. A small dark clot of blood was found protruding from left main bronchus.

Upon slitting up the bronchi it was seen that this clot occupied the centre of a second ulceration, located about one inch below commencement of left bronchus. A small probe passed by side of the clot entered readily one of the pulmonary veins. The blood clot having been withdrawn, the ulcer was found to be 7 x 4 m.m. in size, extending through the thickness of the bronchial wall and communicating with a pulmonary vein by a slit-like opening, 1 x 3 m.m. in size. The wall of the vein was considerably thickened at this point. The tuberculous process commencing in the bronchial mucosa has invaded the entire thickness of bronchus and afterwards the walls of the vein. Several enlarged and caseous bronchial glands are present. This case illustrates the way in which hæmoptysis usually causes the death of very young children in tubercular diseases of the lungs. There are on record very few cases of hæmoptysis in infants, and this is only the second time that I have observed this complication in children. Henschel records his experience as follows: "On the whole, hæmoptysis is extremely rare in children before the period of second dentition, although twelve to fifteen children, from the age of two and one half to five years, have come under my notice who, during severe coughing spells, expectorated small quantities, occasionally a teaspoonful, of blood, either pure or mixed with mucus and pus. In one case the compression or perforation of a branch of a pulmonary artery or vein by cheesy bronchial glands and the simultaneous perforation of a bronchus gave rise to an enormous hæmoptysis." Vogel, in his work, states that he had never observed but one case of pulmonary hæmorrhage and that was in a girl ten years old. Menjer and Repper write that hæmoptysis very rarely occurs in the early stage, or during the progress of the disease; and when it occurs as the cause of sudden death, is due to the complication with bronchial phthisis, rather than to the rupture of a blood-vessel in a pulmonary vomica.

I quote the following from Holt (*Diseases of Infancy and Childhood*): "Hæmoptysis is a rare symptom but not unknown even in young children. Henschel has reported a case of fatal hæmoptysis in a child ten months old, where the hæmorrhage was due to the rupture of an aneurism in the wall of a cavity. Herz, in 247 clinical cases of tuberculosis in children, records eight of hæmoptysis—four of them under five years, and the youngest only eighteen months old. The records of 131 autops-

sies on tuberculous children in the Pendleburg Hospital, show that hæmoptysis was four times a cause of death; two of these patients were under five years, and one was only twelve months old. As in adults fatal hæmoptysis is usually due to the opening of a large vessel by ulceration in the wall of a cavity, which is sometimes in the lung and sometimes in one of the bronchial glands."

There is on record a case of hæmoptysis in a child sixteen days old (*Dict de Med. et de Chirurg.*, vol. xxix.)

A case of death from pulmonary aneurism and hæmoptysis in a child aged two and a half years is reported in the *London Path. Soc. Trans.*, vol. ii; also one by Powell (*Med. Times and Gaz.*, June, 1874), of a child seven months old from a similar cause.

ACUTE NEPHRITIS OF MALARIAL ORIGIN.

BY CHARLES GILMORE KERLEY, M.D.,

Lecturer on Diseases of Children, New York Polyclinic; Assistant Attending Physician, Babies' Hospital; Attending Physician, Out-Patient Department, Babies' Hospital, New York.

When the topic of my paper was sent to the secretary, I hoped to acquire from medical literature, sufficient data, in order that I might furnish a fairly comprehensive article on the subject. In this I was disappointed, having been able to find but meagre reference to *acute* nephritis of malarial origin. The books refer to malaria as an occasional cause of nephritis and go no further. Moncorvo, in an article on malaria in infants, which appeared in 1895, states that in Rio Janeiro acute nephritis is not very rare in children affected with malaria. He claims that it appears far more frequently than is generally supposed, but fails to state the percentage of cases in which the complication occurs. It is his opinion that the malarial poison affects the kidney structure in a manner identical with diphtheria, scarlet fever and other infectious diseases. The literature on hæmaturia, complicating malaria, a very common affection in the southern states, is very unsatisfactory. Whether the cases reported were those of simple hæmaturia, or whether the hæmaturia might be considered one of the manifestations of an acute nephritis, I was unable to determine on account of the lack of observation by the authors.

Recently, Thayer of Baltimore, read a paper on acute nephritis of malarial origin in adults, before the Society of American Physicians, in Washington. He states that 758 cases of malaria treated in the Johns Hopkins Hospital showed albuminuria in 46.4 per cent. Casts of the urinary tubules were found in 17½ per cent. The majority of the cases of albuminuria were observed in the estivo-autumnal fever.

Out of 1,832 cases of nephritis treated in the hospital and out-patient department, 26 were found to be of malarial origin. Of these, 13 recovered; 4 died; and in 9 the results were doubtful. He maintained that nephritis occurs in about 2 per cent. of all the Baltimore malaria cases. Nothing especially distinctive in the clinical character of the disease was noted from that of ordinary toxic nephritis. The anatomical changes in the two cases which

came to autopsy differed in no degree from toxic nephritis due to other causes. He believes that malarial infection, especially in the more tropical countries, plays an appreciable part in the ætiology of chronic renal disease.

The case of which I will give a detailed report, presented the following unusual manifestations:

A girl of eighteen months became ill with fever and vomiting. There was no diarrhœa; the stools were normal; moderate prostration. The urine was noticed by the mother to be of a very dark color. The child had had attacks of indigestion, and the parents looked upon the present illness as a repetition of the attacks. The temperature was 102° at noon the *first* day of illness, but returned to the normal in the evening. The following morning the temperature arose to 103° with increasing prostration and irritability. There was but little desire for food, but no vomiting.

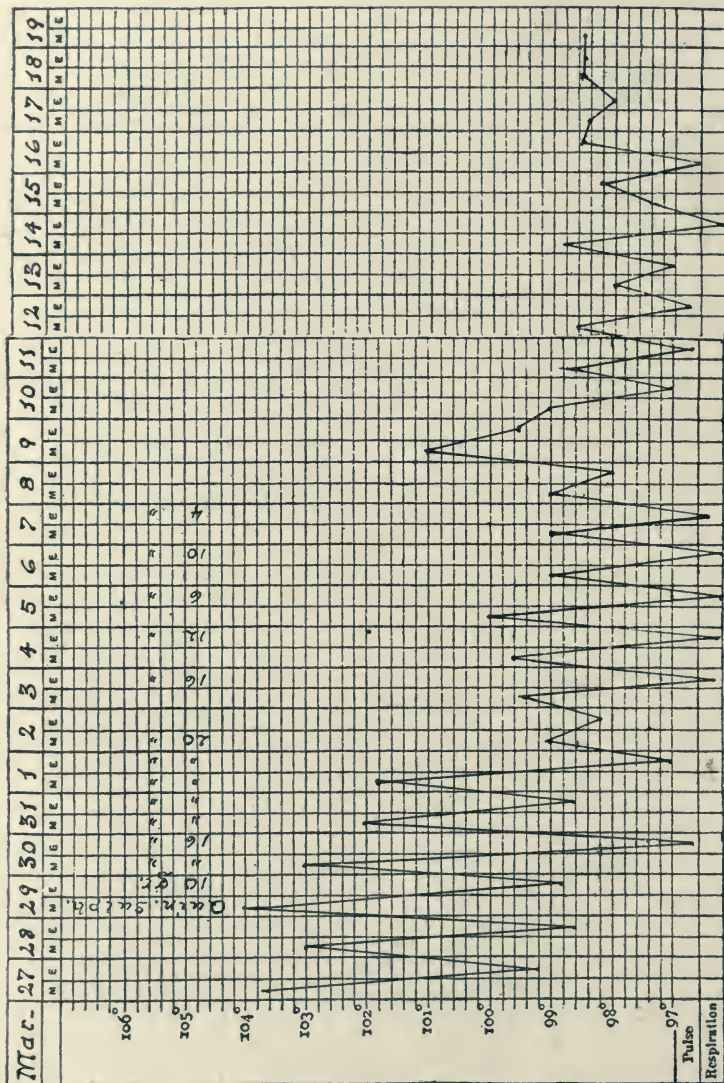
The writer first saw the patient at noon on this, the *second* day of the illness. The temperature at this time was 102° F. There was slight prostration; no vomiting. Considerable milk and barley water in equal quantities had been taken. The urine, which had been saved for examination, was of a bright red color, evidently containing a large amount of free blood. There had been no suppression, the urine having been passed freely. The temperature fell during the afternoon, and at 6 P.M. it was normal. Physical examination of child was negative.

The examination of the urine showed free blood in large amounts, hyaline and granular casts, after filtration 25 per cent. of albumin by volume.

The treatment consisted in rest in bed, diluted milk diet, calomel, one grain in divided doses. At 10 P.M. the temperature began to rise, and reached 104° the next morning at 11 o'clock, the *third* day of the illness. After two such exacerbations and remissions in the temperature, distinctly periodical, a diagnosis of malarial infection was thought to be justified. Ten grains of quinine were given during the third day. The temperature fell to normal in the evening. The urine was unchanged. A quart of milk and barley water was taken in twenty-four hours. Stools were normal, and spleen not enlarged.

The temperature remained normal until 2 o'clock the following morning, the *fourth* day of the illness. At this time it commenced to rise and reached 103° F. at 11 o'clock. The child

was now becoming somewhat anæmic from the loss of blood; the symptoms otherwise unchanged. Sixteen grains of quinine



TEMPERATURE RANGE IN A CASE OF ACUTE NEPHRITIS OF MALARIAL ORIGIN.

given during the day. In the evening at 8 o'clock the temperature was 96.2° F. The pulse was fairly good. The patient suffered no inconvenience from the low temperature, other than cold extremities, which were relieved by hot water bottles. At

midnight the temperature was 97° F.; from this point it arose gradually until at noon on the *fifth* day of the illness it reached 102° F. In the evening it again reached the normal. The urine was passed freely. Examination showed the albumin diminished one-half, but free blood and casts were present. There was no vomiting; no stupor; nourishment well taken. Sixteen grains of quinine were given during the day. The temperature remained normal for a few hours. At midnight it was 102° F., and at 8 A.M., 103.4° F. Quinine, no effect. On the *sixth* day, at 4 P.M., it was again normal, and at 6 P.M. it was 97° F. Sixteen grains of quinine were given. The heart action becoming rather irregular, a condition possibly due to the quinine, it was thought best to give $\frac{3}{16}$ gr. of strychnine three times daily. As there was no enlargement of the spleen, and the quinine having failed to make any impression on the disease, an examination of the blood was made to verify the diagnosis. The specimen was sent Dr. Wollstein, pathologist at the Babies' Hospital. The report was as follows: "Blood specimen shows a number of intra-corpuscular bodies, round and very irregular in shape with light centers and pigment granules at the periphery. The red blood cells containing these organisms are paler and slightly larger than the other red cells. *Diagnosis*—Amœdoid bodies of the tertian malarial parasite."

As will be seen by the accompanying chart, the temperature showed a tendency to a late evening or midnight rise.

In order to use the remedy to the best advantage, seven gr. of quinine muriat were given subcutaneously at 11 P.M. on the sixth day of illness, a short time before the customary rise. The treatment proved effective.

The following day, the *seventh*, the temperature did not rise above 99.2° F. Sixteen grains of the sulphate of quinine were given by the mouth. The urine showed marked improvement, but still contained albumin, 5 per cent. by volume, free blood, and casts. One pint was passed.

On the *eighth*, *ninth* and *tenth* days, the temperature, while not going above the normal, still showed the tendency to the late evening rise and the noon-day fall. On account of the falls to the sub-normal, the quinine was given in smaller quantity, as indicated on the chart, and discontinued on the *thirteenth* day when the urine was free for the first time from albumin and casts. The story of the convalescence is without interest ex-

cepting the tendency of the temperature to the sub-normal. The slight rise on the fourteenth day was due to overfeeding. The child made a satisfactory recovery. She came through the illness unharmed, except a moderate degree of anæmia.

113 WEST EIGHTY-THIRD STREET.

DISCUSSION.

DR. NORTHRUP.—I had the pleasure of seeing this case with Dr. Kerley, and I am very glad indeed to have him report it here, so that those who are situated as I was at that time, having never seen such a case, may be on the lookout for it. I had never seen a case of acute nephritis where the urine was filled with blood and casts, hyaline and granular, that was due to malaria until that time. About the next day there came a little circular from a doctor in Baltimore, in which he stated that he was going to read a paper on nineteen cases of acute nephritis due to malaria. The very next day, the third, I saw another child with the same symptoms, with marked albuminuria, in which the same routine was gone through. The blood was examined and the malaria plasmodium found. This also remained obstinate to quinine by the mouth and yielded to injections, all symptoms disappearing. So far as I now recall, I had never seen a similar case, until Dr. Kerley's.

DR. CAILLÉ—I believe it is generally known that malaria infection may produce nephritis. I have always looked upon malaria as an etiological factor in nephritis. Medical men who come from the Congo districts report such cases as occurring among children as well as among adults, and similar report comes to us from medical missionaries in China. Southern practitioners and physicians from the Mississippi Valley who come to New York, report such cases. If hospital physicians will have a routine examination made of the blood, made in cases of nephritis, they will find malaria frequently a causative factor.

DR. FRUITNIGHT.—I wish to report four cases of this character, three acute and one chronic. The three acute cases suffered from hæmaturia. The chronic case was a boy between nine and ten years old living on Staten Island, who had suffered from recurrent attacks for three or four years. When I saw him he had an acute attack. The malarial plasmodium was found in his blood, and a large number of hyaline and granular casts in the urine. He died in a few days in uræmic coma. One of the other three cases was seen in consultation with the late Professor Loomis. Under quinine, in about a week's time, it was improved. The patient was a boy about eight years of age. The two other cases I saw last fall, one three and the other five years old, and they had typical attacks of quotidian fever, the plasmodium being

found in the blood. The blood was loaded with blood pigment on examination and the children were sick for a very long time, some five or six weeks. They presented the usual cachectic appearance, the skin had that characteristic yellowish-green hue, and they were altogether miserable. As the doctor says, I think malarial infection can frequently cause nephritis, that is if it is unrecognized. When hæmaturia is present as a symptom in any case, we should not omit to examine the patient for a possible malarial infection, for in very many cases of nephritis due to malaria, hæmaturia is very apt to occur at some period in the course of the nephritis.

DR. FORCHHEIMER.—I was astonished by the remark that the literature is meagre on this subject. I have had occasion several times to look up the literature of the subject, especially recently, when I wrote the article in *Keating's Encyclopedia*. Certainly in that article I have made a rather extensive mention of nephritis in malaria, and have described a peculiar form of nephritis, or rather of albuminuria that occurs in connection with malaria in children. At the time a paper on malarial nephritis was read in Washington by Dr. Thayer, I was able to present a specimen of urine which illustrated this special form of albuminuria. It is not the most common form of nephritis, but it is characterized by a black deposit of pigment, the nature of which I have not been able to determine as yet. Of course it can be only one of two things, either some product of hemaglobin, or melanin produced by the plasmodium. The manifestations of malaria present themselves differently at different times and in different places. The form of malaria we have had the last fall and winter in this region has been the estivo-autumnal form. It has been uncommon to find the intermittent form of the tertian or quartan type, although the former has occurred. Depending on the character of the poison and on the individual, depending possibly on the intensity of attack, and depending especially on the chronicity of the attack, we find albuminuria. But albuminuria is only one of the symptoms of nephritis, and the question is where to draw the line. General nephritis, or acute parenchymatous nephritis, or toxic nephritis, as we find in scarlatina, or diphtheria, or measles, is rare in malaria as we see it in this part of the world. In the tropics this form of nephritis is common and adds not a little to the gravity of the attack. I have seen one child in whom a chronic interstitial nephritis was, I think, produced by a chronic malarial affection. But in all the cases I have seen, and they have not been a few, I have not seen the relation established positively between chronic nephritis and chronic malaria. Indeed, if I were forced to a statement, I would have to say that the relationship is one that cannot yet be looked upon as positive.

DR. ADAMS.—Dr. Kerley cannot have looked very carefully into the literature, because in 1880 Dr. Busey read a paper on

chronic parenchymatous nephritis in children due to malaria or malarial poisoning. I helped in the preparation of the paper, being at that time the resident physician in the Children's Hospital, where the observations were made. The title of this paper can be found in the Index Catalogue under nephritis or malaria.

DR. JACOBI.—Only one remark. The fact that a number of observations have been made does not detract certainly from the meritoriousness of the paper, which is very lucid and very instructive. I think many observers may be disappointed in not finding nephritis in malaria when others in other years may find it or have found it. It is a fact which has been observed that in a number of epidemics and endemics of malaria, no nephritis was found, while in another year in the same neighborhood, nephritis was found. Bartels made his observations in Holstein and found it often. Rosenstein, in his classical book on the diseases of the kidneys, says he did find it on the Baltic Sea, but he did not find it in Holland. Heidenheim did not find it for years in a province along the shore of the Baltic Sea, and then again in the very last year he did find it. They all, however, agree that they find much more chronic than acute nephritis, but that acute nephritis does occur has been proven today. Still, as early as 1880, in the first session of the newly established children's section of the American Medical Association in 1880 (Richmond), S. C. Busey reported cases of malaria nephritis observed in infants. The occurrence of amyloid degeneration of the kidneys in chronic malaria is frequent in malaria-stricken countries, such as Algiers or Hungary.

DR. KERLEY.—I spent something like three or four afternoons at the Academy to learn what I could of the literature, and I found a great deal of it unsatisfactory and not at all conclusive. The observations lacked proof either on account of absence of the plasmodium, or absence of enlargement of the spleen. In fact, I did not consider the literature there of any special worth in considering acute toxic nephritis of malarial origin. Acute nephritis of malarial origin is the same as an acute nephritis due to scarlet fever or diphtheria or any of the acute infections. I found of those, very few cases that I considered authentic.

THE SCOPE AND LIMITATIONS OF HOSPITALS FOR INFANTS.

THE PRESIDENTIAL ADDRESS DELIVERED BEFORE THE AMERICAN PEDIATRIC SOCIETY.

BY L. EMMETT HOLT, M.D.,

New York.

GENTLEMEN OF THE AMERICAN PEDIATRIC SOCIETY:—Our meeting this year completes the first decade of the Society. From the small beginning made by a little group in Washington in September, 1888, we have grown to an organization with fifty-four active members, and have done, as a Society, work which has made our name known on both sides of the Atlantic and reflected honor upon American medicine.

In the evolution of this comparatively new specialty, the American Pediatric Society has done much to mould medical opinion. To this body of men, made up largely of the teachers of pediatrics in America, the profession here still looks to work out many of the difficult problems presented by disease in early life. Something we have done, but how little in comparison with the great work yet to be accomplished.

Since our last meeting two of our most distinguished members, both of whom have filled the office of President of the Society, have died. Dr. J. Lewis Smith was one of the pioneers in this department, and his book has probably been more widely read than any book on pediatrics in the English language. Dr. Smith passed away in the fulness of his years, in the midst of the arduous work with which his life had been filled. And how can we express our loss in the death of Dr. Joseph O'Dwyer? Where shall we find another to fill his place? In his modest manner, his rare judgment, his mental balance, and his straightforward method of attacking a single problem and patiently working out its solution to the minutest detail, he was indeed

an example to all of us. We miss his genial face among us to-day, and cannot but feel that our Society has met with an irreparable loss.

And now may I ask your attention for a few minutes to a subject which has for me the deepest interest and which must be of vital importance to every pediatricist, viz:

THE SCOPE AND LIMITATIONS OF HOSPITALS FOR INFANTS.

As the past thirteen years of my life have been intimately connected with such institutions I have thought that nothing which I could bring before you would be of more interest than some of the questions connected with their medical management. It has always been true in the history of medicine that special hospitals have followed the development of new departments, these hospitals being at once a cause and a result of such scientific interest. Europe has had its children's hospitals for nearly half a century, and now almost every continental city may boast of a well equipped one. In America, however, hospitals for children are still few, and in many of them the greater part of the service has been given over to the department of orthopedic surgery or to wards for contagious disease; while little room has been left for general medical cases, and usually none at all for children under two years old. Provision for the hospital treatment of sick infants has been the last to come, but it is coming fast, both in the organization of separate hospitals and in the addition to many of our general hospitals of a ward for infants.

The claim of pediatrics to be recognized as a special department of medicine must rest upon the fact that it is devoted to the problems connected with disease in the first years of life. I venture the prediction that the pediatricist of the future will not be he whose interest lies in whooping-cough, scarlet fever, diphtheria, measles, and other diseases which simply occur more frequently in early life than later, but he who devotes himself to diseases and conditions peculiar to the first three years of life. The hospital of the pediatricist, therefore, is not the hospital for contagious diseases, nor yet one where only children over four years are received, but the hospital for infants and very young children.

Hospitals are needed in this department, in the first place, as places of research. The question of the saving of infant life is fast becoming a vital one in social economics. In New York

City during the past eight years, the mortality of infants under one year has been 86,738, being about one-fourth of the total death-rate, and very nearly the same proportion as is maintained in the cities of Europe. But it is interesting to note that in New York, with increasing knowledge and better sanitation, there has been during the last few years a very decided reduction in this mortality. The year 1897 showed a death-rate under one year, nearly one thousand less than that of any recent year. These are actual mortality figures, it should be remembered, not percentages, and this reduction has been brought about in spite of a steady increase both in the population and in the number of births reported. These facts are certainly most encouraging and should stimulate all of us to do our utmost to improve city sanitation and to spread knowledge upon all subjects relating to infant feeding and care.

But still more encouraging are the results now obtained in private practice among the better classes. Of 151 children who, during the past eight years, have been under my care through practically their entire infancy, not one died before reaching the end of the second year. This is still more significant when we consider how they were fed: only thirty of the number were breast-fed either by the mother or wet-nurses through the greater part of the first year; thirty-three were partly nursed and partly fed, nursing being usually continued for two or three months alone, after which the bottle was added; ninety were entirely bottle-fed. This certainly makes a good showing for artificial feeding and indicates very clearly that among the class of people where artificial feeding can be properly done, it is extremely satisfactory.

During the past eight years while I have been practicing almost exclusively among children, I have had among my own patients but six deaths in children under two years; one from marasmus and a cerebral malformation; one from acute inanition, under observation but ten days; one each from general tuberculosis, intussusception, entero-colitis and pneumonia. I do not think these are exceptional results; for on inquiry I have learned from six professional friends in New York, all of whom practice among children, that their experience in the same class of patients was almost identical with my own. From the facts thus collected I judge that in the well-to-do classes, with the best care, the mortality from all causes during infancy does not exceed two

or three per cent., as against a general mortality for this period among all classes of about twenty per cent. These are most hopeful signs and show the possibility of a very great reduction in infant mortality everywhere with a better understanding of all the conditions, but especially of infant feeding.

Referring again to the death-rate in the city of New York, we find that 34 per cent. of the entire number of deaths occur in children under two years, and only 12 per cent. in children from two to fifteen years. Or, in other words, as it is well known that morbidity and mortality figures correspond very nearly, three times as much serious sickness occurs in infants under two years as among all the other patients coming under the care of the specialist. Conditions hereafter to be considered make it undesirable, and in fact forever impossible, that any large percentage of these shall be treated in hospitals, although there are those who look forward to the time when most adult patients shall be treated in institutions, both obstetric, medical and surgical cases. But it is none the less true that only in hospitals can any great headway be made in the solution of many of the problems connected with infantile disease. Let it be clearly understood then from the outset, that hospitals for infants serve perhaps their highest function when they can determine from careful study and observation of the few what is the best treatment for the many.

As places for research, hospitals must be well equipped with pathological, bacteriological and, if possible, chemical laboratories, in order to work out in the fullest and best way the problems constantly arising in the treatment of acute illness. The state spends without grudging large sums of money every year in the experiment stations of the agricultural department to determine the best conditions under which hogs, cattle, fruits, etc., shall be raised; why shall it not devote at least as much of its energy toward the solution of the problem how infants may best be reared, and how the great sacrifice of infant life which now exists may be diminished.

Hospitals are needed, in the second place, for the teaching of physicians and students. One of the greatest deficiencies in the curriculum of the medical schools of the day is the insignificant attention paid to subjects connected with infancy. In order to give students an opportunity for study, hospitals are absolutely necessary. Every hospital should be a teaching hospital. This

work in no way interferes with its function of caring for the sick, and has a far wider value in philanthropy than the caring for the children in the wards. The selfishness of those hospital physicians in America who are content to enjoy for themselves the peculiar privileges and opportunities which their positions carry with them, with no thought of their obligation to advance the science of medicine, is unworthy of our profession.

Thirdly, hospitals are needed for the training of nurses. It is just as impossible for nurses as for physicians to learn how to take care of sick infants in the wards of a general hospital, and trained nurses are quite as important for the well-being of the public at large as are trained physicians.

Fourthly, hospitals are needed for the care of such cases as can be better treated in institutions than at home. Of this more will be said later.

The question naturally arises whether a department for infants in a general hospital may not be more advantageous than an institution specially established for this work. My own belief is in favor of special hospitals for the following reasons: It is hard to rouse in the average house physician proper interest in the nutrition and diseases of infancy while he sees around him on every side the brilliant counter-attractions of surgical work and an acute medical service among adults. It is only after the young physician has begun his private practice that he appreciates the value of a knowledge of diseases of infants. Again, it is usually the case that the attending physicians pass over the ward for infants with very scant attention. This may be remedied, of course, and always should be by the appointment of a special attending physician to these wards. But perhaps the chief difficulty is with hospital boards; it being difficult or impossible to make them appreciate the fact that the requirements in the hospital treatment of infants are very different from those for adults.

Hospital work for infants has its discouraging features; the first years of every such institution are sure to reveal many of these. One of the most prominent is the very high mortality—something which was not previously expected and which, as compared with the mortality of ordinary adult hospitals, seems simply awful. This has often so discouraged boards of managers as almost to induce them to give up the enterprise altogether. It is difficult to determine what a reasonable mortality in hospi-

tals for infants should be on account of the many different conditions which affect different institutions. It must be remembered that the mortality of infants under one year is high in all cities, even outside of hospitals; and that in institutions where chiefly cases of serious illness are brought, it must of necessity be very high. Of 1,217 cases under one year old admitted to the Babies' Hospital in seven years, there were 548 deaths, a mortality of forty-five per cent; and if we should add those removed by friends when a fatal result was inevitable, in order that they might have the consolation of the child's dying at home, the mortality would run up fully fifty per cent. The reasons for this high mortality are to be found in a study of the class who make up the bulk of patients who are sent to a hospital for infants. These are:

1. Marasmus cases, the majority being under six months old; they are children whose mothers are dead, or sick, in hospitals, in asylums, or intemperate. Often they are the children of wet-nurses who have been boarded out. Whatever their origin they have been neglected and badly fed, and have gone steadily down until as a last resort they are sent to a hospital.

2. Cases of acute starvation; usually infants under three months old; often on account of extreme poverty or destitution they have had no shelter and no food but tea or beer for days. Many of these at last find refuge in a hospital and live a few days or perhaps but a few hours.

3. Cases of neglect where systematic and regular drugging has been continued, usually by opium in some form.

4. Cases of acute pulmonary or intestinal disease which have been some time sick and have grown alarmingly worse at home. These are sometimes brought by friends as a last resort and sometimes sent in by physicians when they see that a fatal result is probable.

5. Cases of disease which are almost certainly fatal, such as tuberculosis, all forms of acute meningitis, hydrocephalus, serious malformations like those of the intestines, intussusception, etc.

Regarding cases which are hopeless on admission, like most of those just enumerated, one must admit that they have the same right to hospital care and shelter as have cases of cancer or advanced tuberculosis in adults. They are going to terminate fatally whether they are admitted to a hospital or not, but

no institution which claims to be charitable or philanthropic can refuse to take its share of these hopeless cases. However, the fact that their proportion is so large, is what raises the mortality figures of an infants' hospital so high. But to judge of the usefulness of an institution by its death-rate where such material is received, is manifestly unfair; nor can it be in any way compared with the mortality in hospitals for adults.

Another discouraging factor to many hospital managers is the expense required. This consists largely in the care and nursing, since the cost of food and supplies play but a minor part. Our lowest expense in the Babies' Hospital has been about one dollar and twenty cents a day *per capita*, and as the work has been better done the expense has risen until it is now about a dollar and a half per day. It is hard to make hospital managers appreciate the fact that the value of work consists not in its amount but in its quality.

There are some peculiar conditions in infants' hospitals which must be considered; those which affect the nutrition of the patients are of the highest importance in modifying the results obtained and are in fact paramount to every other consideration. In private practice the outcome of every acute illness among infants is determined chiefly by two factors. One of these is the patient's previous nutrition. While we do see infants, previously robust, who succumb to acute attacks of pneumonia or intestinal disease, by far the greater number of those who die, perish because they were previously feeble or delicate. The second factor is how well the patient's nutrition can be maintained during the acute attack. This relates not merely to food and feeding, but to all conditions affecting the nutrition—air, bathing, clothing, and general care. It often happens that the disturbance of the patient's general nutrition is more profound and more serious than the local effects of the disease. Thus, a child may have an acute attack of pneumonia which runs its usual course; the lungs clear up; the cough disappears; but the infant does not recover his previous health. Before this disturbance he was sufficiently vigorous not only to maintain existence but to increase in weight; now, however, the balance is against him and he cannot seem to make progress no matter what is done. Little by little he continues to lose in weight until he finally dies from marasmus. Not only do these results follow serious acute diseases but attacks of bronchitis, or indigestion,

which are not very severe and sometimes such a mild thing as tonsillitis.

Another striking thing seen in hospitals is the frequency with which one acute attack is followed by a second, and perhaps this by a third, until the child is finally worn out. I have seen as many as six distinct attacks of pneumonia in the course of three or four months—the lungs between the attacks in some instances clearing entirely, as shown by the signs, and in others great improvement taking place but never quite the evidences of local recovery. To be sure, one sometimes sees such things in private practice, but they are seldom met with unless hospital conditions are approached, both as to surroundings and the previous condition of the patients.

In adults, prognosis in most acute infections depends upon the severity of the attack and the integrity of the important viscera—the kidneys, heart, liver, etc.; but in infants, upon the nutrition of patient, even more than upon the severity of the attack, since in a child with feeble resistance even the slightest attack may begin processes which go on to a fatal termination.

We come therefore to the conclusion that the first condition of success in hospitals for infants is a solution of this problem of nutrition, difficult in health often even under the most favorable surroundings, much more difficult in disease, but in disease and under unfavorable surroundings well-nigh insoluble. The things to be considered as affecting nutrition are mainly four: air space, ventilation and airing, nursing and care, feeding.

THE AIR SPACE.—In most hospitals for infants the maximum allowance is only 600 or 700 cubic feet to each bed. At the Randall's Island Hospital, New York, it is in many wards much less than this; in one of their diphtheria wards it was but 400 cubic feet. At the Nursery and Child's Hospital, 650 cubic feet has been set by the Board of Health as the allowance. At the Babies' Hospital we began with this space but found it to be totally inadequate. For the last three years the allowance per bed has been 800 cubic feet, and it is now 1,000 cubic feet. With less than this I am sure the results will always be unsatisfactory with children under one year. For those who are beyond this age a little less may suffice except in cases of very acute illness. Our experience may be stated somewhat as follows: two infants in a ward of a given size do well; three may do fairly; four are sure to do badly, and, if the number is

increased beyond this point all will fail rapidly and some will soon die. In the old Emigrant Hospital on Ward's Island, Dr. A. M. Thomas found that infant feeding could be conducted almost as satisfactorily in the ward of an institution as in private practice, but his experiment was conducted with an allowance of 2,500 cubic feet to each child.

The question of ventilation is a difficult one and it must be considered with that of airing infants. No matter how good the ventilation of the ward, infants must be taken from it once or twice a day to an apartment where an entire change of atmosphere is secured. Separate rooms for airing, protected balconies or sun-gardens must form a part of the equipment of every hospital if good results are to be obtained; some provision, however, must be made to get infants out of their habitual atmosphere. The youngest, and those most acutely ill, need it most, and should have a change of at least two or three hours a day, thorough airing and ventilation of the wards they have left being accomplished meanwhile. Nothing is quite so fatal as overcrowding in close, superheated rooms. The time of airing and the change of temperature allowed should be carefully regulated by the physician and made a distinct part of the child's treatment. In the wards, ventilation should be as perfect as possible, and there should be open fires whenever these can be secured. These things are often carefully looked after during the day, but entirely neglected at night.

Cleanliness in its minutest details should be secured at whatever cost. Under the artificial conditions of hospital life purity of the atmosphere is absolutely necessary. Nothing which contaminates the air of the ward should be permitted. Soiled napkins should be immediately removed. No gas should be used at night; but, if not electricity, the wax night-lamp of the nursery. Frequent fumigation of the rooms with formaline or sulphur should be practiced where cases of acute illness are continually treated. The floors should be wiped daily with cloths wrung out of antiseptic solutions. All of these matters, of course, greatly increase the labor of hospital work, and consequently the expense. It costs to keep clean, but the surgeons have demonstrated to us that it pays. The essential hygienic conditions belonging to a well-appointed nursery are indispensable to good hospital work.

The temperature of the different wards should be suited to

the nature of the cases treated. Thus 75° F. may be necessary for premature, very feeble and some marantic infants; 68° to 70° F. for ordinary cases of acute illness; 66° to 68° F. for those not acutely ill and for most children over one year old. For convalescent children provision should be made for additional outings on balconies, piazzas, and in hammocks and perambulators about the grounds whenever the weather will permit.

NURSING AND CARE are no less important than the matters already mentioned. Infants require fully four times as much care as any other class of hospital patients. No good results can be expected where a ward with twenty babies is given into the care of three or four nurses, however intelligent and industrious. It has been my experience that even in the case of infants who are not acutely ill, not more than three can be well cared for by a single nurse, and for those who are seriously ill one nurse to every two children is the minimum. This nursing must be constant, and it does not consist simply in the administration of food and medicines, and the application of clean diapers; infants must be taken from their beds, held, given opportunities for change of position, and in most cases for a certain amount of exercise.

FEEDING.—To obtain the best results a hospital for infants should have command of every method of feeding; not only the most approved methods of artificial feeding such as can be furnished by a milk-laboratory in the hospital, but with wet-nurses for certain special cases. Nothing is more discouraging than the great difficulties in the way of artificial feeding in institutions of children under six months old. For those wasted specimens admitted with chronic indigestion and malnutrition practically nothing can be done by any method of artificial feeding which I have ever tried.

The greatest difficulties in the hospital's treatment of infants are met with in patients under one year. After this age the conditions of nutrition are usually not so hard to control, and the proportion of those children who do well is very much larger. The dangers of "hospitalism" are therefore inversely proportionate to the age of the patient. In a very young infant whose organism has not yet acquired stability, resistance is so feeble that it can not long be subjected to hospital conditions without serious injury. In such patients it seems to be impossible to combat at the same time the effects of acute disease and a depre-

ciated nutrition. But with every month of advancing age the problem becomes easier, and when their nutrition may be said to be established, they can withstand the deleterious influences of hospital life, and most diseases can be successfully treated.

According to my experience, the diseases and conditions during the first year which are especially suited to hospital treatment are acute pneumonia, empyema, acute forms of gastrointestinal disease, otitis and its complications, ophthalmia, acute surgical cases and most cases of eczema in children over six months old. In all acute diseases it is unwise to retain the infant in the hospital after the acute stage of the disease is past unless the hospital is in the country; and even here in a few weeks the baneful effects of hospitalism are frequently apparent. The retention during the whole period of convalescence is fraught with great danger and is very frequently followed by secondary attacks. In a large number of the cases above referred to the condition is somewhat like this: The disease can be cured provided the child is sufficiently strong and old enough to stand the strain of life in a hospital. This is a question to be considered in every disease which requires a prolonged stay. One of the most striking limitations which one encounters in the treatment of infants in hospitals is in the management of cases of chronic nutritive disorders. Complete restoration to health and vigor of such children in a hospital, although not impossible, is not to be expected. Perhaps the most that can be done for them is to keep them long enough to decide the question of the method of feeding best suited to the case, and then care for them as out-patients.

During the second and third years the results in the treatment of all diseases are very much more satisfactory. Not only may all the diseases enumerated in speaking of the first year be successfully managed, but also many chronic nutritive disturbances, such as simple malnutrition, chronic indigestion, rickets, scurvy, cretinism, some chronic surgical cases, and under certain circumstances some of the contagious diseases, provided the hospital has facilities for their isolation. With reference to the admission of contagious diseases during infancy, however, great dangers and very bad results are usually seen from grouping many children together, particularly in measles, diphtheria and whooping-cough, where the tendency is so great to the development of broncho-pneumonia amid such surroundings. Many of the bad results attributed to the use of antitoxin in infants are

simply the effects of hospitalism and were seen years before antitoxin was known. In a recent epidemic of measles seen in the Nursery and Child's Hospital, over one-third of all the cases were complicated with pneumonia, and in some wards almost every child with measles developed pneumonia. Dr. Crandall tells me that of thirteen cases of measles in one crowded ward in the Randall's Island Hospital, twelve were fatal, all from pneumonia. Results in measles and diphtheria during the first two years are, I believe, much worse in crowded hospitals than when children are treated separately in tenement houses, even under very bad surroundings. One of the reasons why cases like those mentioned above are not suited to hospital treatment is the prolonged stay which convalescence from infectious diseases requires. Under such conditions, complications like late pneumonia, entero-colitis, etc., are almost certain to develop, and to these many patients, who escape acute disease, succumb.

There are several peculiar phases of disease seen in infant hospitals. One of the most striking of these is hospital marasmus. While it occurs most frequently in children already suffering from malnutrition, or in those who have previously suffered from acute disease, it is seen in others who were perfectly well on admission. After gaining perhaps for a week or two, such children, as a result of hospital surroundings, begin to lose appetite and color; they show stationary weight, then a loss of a few ounces; then vomiting begins, and a more rapid loss continues, until a condition of marasmus is reached. Some of these children show no evident signs of indigestion, and may continue to have good, well-digested movements from the bowels. Gradually they become more and more anæmic, frequently there is general œdema, and at last they succumb to marasmus. Toward the end there may be an intercurrent attack of some acute process like bronchitis, acute diarrhœa, possibly broncho-pneumonia; but death often occurs without any of these, the autopsy showing no lesions to explain the fatal result. Hospital marasmus is rarely seen except in infants under eight months old, and occurs most frequently in those under four months old. It demonstrates beyond any question how injurious to infant life is the atmosphere of a hospital. As already stated above, one-third of the deaths in children under one year old who are admitted have been, in my experience in the Babies' Hospital, due to marasmus.

A most distressing thing is the frequency with which children admitted for simple malnutrition or some slight ailment, develop some serious forms of acute disease while in a hospital. I need not dwell upon how often the contagious diseases are contracted amid such surroundings; this is well known and fully appreciated. I wish especially to refer now to other diseases, particularly pneumonia and acute intestinal diseases. The hospital records show that during the last ten years no less than thirty children, admitted for minor ailments, developed primary pneumonia, and many more developed inflammation of the gastro-intestinal tract. While without doubt the opportunities of infection from without are very great in a hospital, it is my belief that most of these attacks are to be regarded as examples of auto-infection, and are due to the fact that the child is placed under conditions which greatly diminish his resistance. Few things are more discouraging than to see a child, admitted in tolerably good condition, develop, after a stay of a week or two in the hospital, an acute pneumonia or entero-colitis which proves fatal.

From what has been said, the inference may be drawn that infants should not be sent to hospitals for minor ailments and kept there a long time, also that the effect of combining sick and well children in the same ward has a most injurious effect upon the latter; and, further, that after every form of acute illness, children should be removed as soon as possible from the hospital atmosphere.

The ideal infants' hospital, in my opinion, is not a large one. The general hospital with its two or three hundred beds can not be imitated here. The greater the degree to which infants are massed and crowded together, the more unnatural are the conditions under which they are placed, the worse are the results of treatment and the less valuable does the study of disease under such conditions become as a guide to practice elsewhere. As an illustration of this point, let one study the statistics of the large foundling asylums of Europe. A hospital of fifty or sixty beds is, in my opinion, greatly to be preferred to one of double or treble the size; but it should be provided with every appliance known for the relief of sick infants, and its work should be done under the most approved conditions for achieving the best results. Its hygienic surroundings should be more carefully considered and watched than those of a well-appointed nursery.

Small wards containing from four to six patients are greatly to be preferred to those of fifteen or twenty, if for no other reason than to avoid the danger of contracting contagious disease. So far as work in large cities like New York is concerned, hospital work during the summer should be conducted in the country. With diarrhœal diseases, I believe, the results obtained in close hospital wards in the city are quite as bad as in the tenements.

A reduction in hospital mortality is to be secured not simply by improving our methods of feeding, although this has been the aspect of the subject which has so far attracted most attention, I regret to say, to the neglect of the other phases of the question. This is not all the problem nor, to my mind, even the most important feature of it. Between ordinary methods of feeding and the best methods the difference in results in institutions is not great, indicating very clearly that this is not the factor of chief importance. It is a familiar experience to see an infant, who, in a hospital, has been losing in weight and showing all the signs of perverted nutrition, begin to improve at once and gain steadily when removed from the hospital ward, sometimes upon the identical food which it was receiving, and not infrequently when both the food and the manner of feeding have been greatly inferior. In hospitals not only the feeding, but all other conditions mentioned must receive due consideration as well—the air space, ventilation, airing, temperature, nursing and care. When all these are fulfilled, I believe that the results obtained will fully justify the existence of separate hospitals for infants in bringing about a more accurate and wider knowledge of infantile diseases and their treatment.

Hospitals for infants are necessary, as I have already shown, but they must be conducted with a very definite knowledge of what can be done in them and what should not be attempted. By recognizing fully their limitations, as well as their advantages, they may be made of the greatest possible benefit to the profession and the public. The conditions of success are difficult and the discouragements are many. Those with experience will not say, I am sure, that I have over-stated the facts. The work must be well done to be of any value either to the patients treated or to medical science, and unless circumstances will admit of its being done well it should not be attempted at all.

REMARKS ON THE CLASSIFICATION OF THE ANÆMIAS
OF INFANCY, WITH A REPORT OF A SEVERE CASE.

BY JOHN LOVETT MÖRSE, A. M., M. D.,

Physician to Out-Patients at the City Hospital and at the Infants' Hospital,
Boston. Assistant in Clinical Medicine, Harvard Medical School.

Alice C., ten months old, was brought to the Infants' Hospital July 14, 1897. The parents were Canadians and well. Of eleven older children, seven had died of various troubles; the remainder were well. There was no tubercular or syphilitic history.

She was healthy at birth. She was never nursed, but was fed on various "infant foods." She never thrived, and gained little or no weight. During the last few weeks she had rapidly lost both weight and strength. She had always vomited a good deal, less recently. Had considerable diarrhœa for first few months; since then moderate constipation, the movements being normal in color. Always subject to cough; more so lately. Said to have had pneumonia at eight months. Bunches were noticed in the neck and groins at five months. They had not increased markedly since. Depression of fontanelle, dryness of skin and nocturnal fever had been present for three months. A tumor in the abdomen was first noted a week before she came to the hospital. It had increased somewhat in size during this time. There had been no internal or subcutaneous hemorrhages.

PHYSICAL EXAMINATION.—Poorly developed and emaciated. Skin dry and generally brownish, but pigmentation nowhere localized. No subcutaneous hemorrhages. Frontal bones at a considerably lower level than parietal. Parietal eminences large. Fontanelle depressed, but small. No teeth. Tonsils normal. Whitish bunch, size of a small pea, in median line at junction of hard and soft palate. Slight rosary. Heart and lungs normal. Thyroid not enlarged. Dulness under upper part of sternum. No dulness at root of lungs. Numerous small glands both anterior and posterior to sterno-mastoid muscles. Glands in axillæ, size of pea. Glands size of peas and walnuts in both groins.

Liver flatness above at sixth rib; edge felt three fingers' breadth below costal border. Edge of liver sharp and firm. Spleen felt running from left anterior superior spine across to umbilicus and thence upward under ribs in left nipple line. Notch a little above level of umbilicus. Edge smooth and hard. Slight enlargement of epiphyses.



SEVERE ANÆMIA WITH SPLENIC TUMOR.

Blood. Hæmoglobin,	- - - -	60 per cent.
Red corpuscles,	- - -	4,340,000
White corpuscles,	- - -	31,500

Owing to poor straining it was impossible to make a differential count of the white corpuscles at this time and the red cells were alone examined. They showed very marked variation in size. Poikilocytosis was extreme. Nucleated forms were very numerous. Of 100 nucleated cells, 89 were macroblasts and 11 normoblasts. Six of the macroblasts showed nuclear figures. Many of the nucleated, and some of the non-nucleated forms, were polychromatophilic.

A differential count of the white corpuscles was made from cover-slips prepared five days later, July 19, 1897. This resulted as follows:

Lymphocytes,	-	-	-	-	29.5 per cent.
Large mononuclear,	-	-	-		9.9 " "
Polynuclear neutrophiles,	-	-			56.5 " "
Eosinophiles,	-	-	-	-	.3 " "
Myelocytes,	-	-	-	-	3.8 " "

Strangely enough, nucleated red corpuscles were very much less numerous than at the preceding examination. While counting 1,000 white corpuscles, 17 nucleated red cells were seen, all macrocytes. Three of these showed nuclear figures. Polychromatophilia was much less marked.

The diet was regulated, and small doses of Fowler's solution ordered.

The child was not seen again until August 9th. The general condition was then somewhat better. The physical examination was unchanged, except that the spleen had diminished considerably in size. (See photograph). The blood was unfortunately not examined. The child was not seen again, but the mother wrote in answer to an inquiry, that it died on September 18th. There was hemorrhage from the bowels for two weeks before death; and the "bunch in the side could not be felt."

Before attempting to classify this case it may be well to consider briefly the peculiarities of the blood in infancy both in health and disease. The blood of infants under two years, normally differs in certain of its characteristics from that of adults. The hæmoglobin, although relatively high for a few weeks after birth, is, during the rest of childhood, relatively low. The number of red corpuscles is about the same or a little larger than in adults, averaging a little over 5,000,000 per cubic millimetre. During the first weeks of life there is more or less variation in the size and shape of the red cells, and nucleated forms are not very unusual. The number of white corpuscles per cubic millimetre is somewhat larger than in adults, averaging from 10,000 to 12,000. The relative proportions of the various forms of leucocytes are also considerably different. The limits, as given by Gundobin, are as follows:

Small mononuclear,	-	-			50 per cent. to 70 per cent.
Large mononuclear,	-	-	-	6	" " " 14 " "

Polynuclear neutrophiles, - - 28 per cent. to 40 per cent
 Eosinophiles, - - - - 1 " " " 10 " "

That is, the proportion of mononuclear forms is about three times as great as in adult life, while that of the polynuclear neutrophiles only half as large. The mononuclear cells, moreover, are not merely lymphocytes, but vary much, not only in the size of the cell as a whole, but also in the size of the nucleus and in the amount of protoplasm. Finally, an increase in the number of eosinophilic cells, even if considerable, seems to be of less significance.

Blood changes develop more easily and more frequently as the result of various morbid conditions and diseases in children than in adults. All the changes seen in the blood of adults as the result of disease, are exaggerated in infancy. The tendency is always to revert to a younger or to the fœtal type of blood. All forms of blood disease in infancy are apt to be associated with splenic enlargement.

As the result of this tendency to exaggeration of changes and to reversion to a younger type, the red corpuscles show much greater variation in size and shape and many more nucleated forms than in similar pathological conditions in adults. Leucocytosis also develops more rapidly and to a greater degree. Its type, moreover, is not constant as in adults in whom the increase of white cells is almost entirely in the polynuclear neutrophiles. In children the leucocytosis is sometimes due to the increase of lymphocytes, sometimes to that of the large mononuclear forms, sometimes to that of the polynuclear neutrophiles and sometimes even to that of the eosinophiles. The general tendency, however, seems to be toward lymphocytosis, *i.e.*, toward the infantile type. This variation in the character of the leucocytosis is difficult to explain, except on the assumption that the proportions of the leucocytes correspond to certain tissue conditions and alterations which are at present unknown. The lymphocytes seem, however, to be especially increased in affections of the gastro-enteric tract. Leucocytosis is more apt to occur in blood conditions associated with splenic enlargement than in those without it. Myelocytes occur in less severe conditions and in greater numbers than in adults. The percentage of hæmoglobin is almost always relatively low.

Owing to the peculiarities just noted, the classification of the anæmias of children is at best a vexed one. They, as those of

adults, may be roughly divided into the primary and secondary, the primary being subdivided into chlorosis and pernicious anæmia. Chlorosis, with its typical symptom complex and blood, is not a disease of early childhood. Cases of progressive pernicious anæmia have been reported in young children, but they are all open to criticism, either because they do not correspond to the blood-type accepted for this disease, or because they are secondary. There is no evident reason, however, why this disease should not occur in infancy. In fact, the instability of the blood at this age would seem to predispose to its occurrence. The secondary anæmias may be divided, in accordance with the classification of Monti, into the mild and severe forms—anæmia levis and anæmia gravis. In the former the diminution in the specific gravity, hæmoglobin and number of red corpuscles is slight and the red corpuscles show no histological changes. In the latter the diminution in the specific gravity, hæmoglobin and number of red corpuscles is marked and the histological changes in the red cells are often considerable. In this form marked variation in the size, shape and staining qualities may be present and nucleated forms of all sizes and shapes are not infrequent. Either form may or may not be accompanied by leucocytosis. In all forms, whether or not accompanied by leucocytosis, the spleen may be enlarged. It is evident, moreover, from the above description, that no sharp distinction can be drawn between the various forms, but that all must merge into one another.

It is to the severe form of anæmia without leucocytosis associated with splenic tumor that the term "splenic anæmia" has been applied. There is no justification, however, for setting these cases apart as a special form of disease and giving them a special name, for there is nothing characteristic in the blood condition; the same condition of the blood occurs unassociated with splenic tumor and in connection with leucocytosis; splenic tumor occurs with other blood conditions and with a normal condition of the blood; both blood changes and splenic tumor occur together, as secondary manifestations, in the course of other diseases, notably, rickets. The splenic tumor must, therefore, be regarded either as an accidental association or as a result of the same cause as the anæmia.

Certain cases of severe anæmia associated with marked leucocytosis and with large splenic tumor have been described under the term "anæmia infantum pseudo-leukæmica." There seems to be

no unanimity of opinion among those who use this term, however, as to what constitutes the disease. Von Jaksch, who gave it its name, describes it as a form of anæmia of children whose symptoms and clinical course correspond to the picture of leukæmia. There is marked enlargement of the spleen, liver and glands, a very considerable leucocytosis, but no evidences of leukæmia on section. The course is less rapid and the prognosis better than in leukæmia. The increase in size of the liver and spleen is not proportionate as in leukæmia, that of the liver being relatively less than that of the spleen. The edge of the liver is sharp, not rounded, as in leukæmia. His examinations of the blood are unsatisfactory, and he gives no differential count of the white corpuscles. Luzet and Alt and Weiss describe the blood condition as follows: Constant diminution in the number of erythrocytes; constant more or less marked diminution in hæmoglobin; poikilocytosis; very many nucleated red cells, mostly of abnormal form and many showing karyokinetic figures; polychromatophilia of the nucleated red cells and many of the non-nucleated; pretty marked leucocytosis, always polymorphous. In addition there is always splenic tumor and more or less enlargement of the liver. They apparently attribute no diagnostic importance to the presence or absence of myelocytes. This description is the one which is at present the more generally accepted. Those who consider this blood condition characteristic are divided, however, as to the limitations of the disease. Some, who consider it always primary, would rule out those cases in which it develops secondary to rickets, syphilis or other anæmias, while others would include them. Careful comparison of the clinical histories and blood examinations of the cases reported as examples of this disease show still further discrepancies in the conception of the disease.

There is nothing characteristic in the changes described in the red corpuscles. They may be seen in any severe anæmia. In the same way there is nothing characteristic in the leucocytosis; it is merely a question of degree. Fischl has found the same type of blood in cases of rickets both with and without splenic enlargement. Moreover, cases of progressive anæmia with enlarged liver and spleen do not always show this condition of the blood. In addition, ordinary secondary anæmias have been seen to develop this blood-type while under observation. Von Jaksch claims to have seen it change to a leukæmia, and Muller to a per-

nicious anæmia. These last observations must be considered as doubtful, however. It is evident, therefore, that there is nothing in the etiology, clinical history or blood of the so-called "anæmia infantum pseudo-leukæmica" to justify its acceptance as an independent disease. It is rather to be regarded merely as a severe type of secondary anæmia which may arise as the result of many diseased conditions. Its special peculiarities, marked leucocytosis and large excess of nucleated red corpuscles, are in no way characteristic, but merely due to the age of the patients. While these cases are not sufficiently characteristic to be considered as examples of a separate disease, they are of importance in that they emphasize not only the heterogeneous character of the anæmias of infancy, but also the difficulties in the way of their classification.

In the light of our present knowledge, therefore, a very simple classification is alone justifiable. The following modification of Monti's is, I think, a fairly satisfactory one:

SECONDARY.—Mild anæmia.

Mild anæmia with leucocytosis.

Severe anæmia.

Severe anæmia with leucocytosis.

PRIMARY.—Pernicious.

Leukæmia.

In all of these forms there may be greater or less splenic enlargement. Splenic tumor, therefore, is of little or no aid in the differential diagnosis of the anæmias of infancy. Under the head of severe anæmia with leucocytosis must for the present be included all those anomalous cases which do not correspond to the type of either pernicious anæmia or leukæmia. That is, this class includes the cases which have hitherto been described as "anæmia infantum pseudo-leukæmica." Unfortunately there is practically no data as to the type of blood of either pernicious anæmia or leukæmia in infancy. For the present, therefore, they must be considered to be the same as in adults.

My case illustrates very well the difficulties in the way of classification. The histological changes in the red corpuscles and the great preponderance of large nucleated forms suggest pernicious anæmia. The diminution in the number of red cells is slight, however, and much less than that which usually occurs in this condition. Splenic enlargement to the degree present in

this instance is at least unusual. The number of white corpuscles and their relative proportions is not, however, inconsistent. The enlargement of the liver, spleen and glands suggests leukæmia. The increase in the number of leucocytes is only moderate, however, being much less than that which usually occurs in leukæmia. The percentage of myelocytes, moreover, is small, being no larger than that which is often met with in severe secondary anæmia of any sort. The clinical history and blood correspond pretty closely to those of "anæmia infantum pseudo-leukæmica," as described by Luzet and Alt and Weiss. This condition, however, as already stated, cannot be considered as a disease *sui generis*, but merely as a severe form of secondary anæmia with leucocytosis. My case, too, must therefore be regarded, I think, simply as an example of this large and irregular class of severe secondary anæmia with leucocytosis. The cause of the anæmia in this instance is undoubtedly to be sought in the general malnutrition resulting from improper food. The splenic tumor must be regarded as merely a coincidence, a result of the same cause. That it is not an essential feature of the case is shown by the fact that it became smaller as the case progressed, probably finally disappearing entirely.

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DISCUSSION.

DR. JACOBI.—I am delighted to find that a gentleman who has been studying the blood for years and who is intimately acquainted with it in all its phases, should feel obliged to protest against the multiplication of names and diagnoses. The result of his studies has led to the conclusion that the names we now possess are ample for making our diagnosis, always taking into consideration that the gradual transition and mutability of several forms of anæmias are well established and may occur. We do know, for instance, that cases at one time recognized as pseudo-leukæmia, may afterward turn into leucocythæmia, etc. If I understood correctly, there was in this case enlargement of the

spleen and liver in the beginning. It is not necessary that those be a part of the disease; they may have existed before. The spleen got a little smaller after a while and it appears that is not the case in leucocythæmia. There were 4,100,000 red blood cells and 30,000 white, that is a proportion of 1 to nearly 140, which comes near the boundary line of leukæmia. Afterwards no other examination of the blood was made. The doctor states that there was a poikilocytosis, so it seems to me the case should be claimed to be one of so-called pernicious anæmia. The lymph bodies were swollen all over. That with the diminution in the blood cells would give us the appearance of pseudo-leukæmia, Hodgkins' disease. I understand that a *post-mortem* was not made. If it were not for the poikilocytosis in the case I would say that it was a case of Hodgkins' disease, but the poikilocytosis characterizes it as a pernicious anæmia. There is no reason why these two conditions should not occur at the same time, under the same or similar or different unknown circumstances.

DR. FREEMAN.—I had a case a year ago in which the appearance of the child was much like this. There was considerable emaciation, enlargement of the liver and spleen, considerable anæmia with leucocytosis. The diagnosis was not made until autopsy, when we found an abscess of the left kidney, which had not caused a large tumor. It had produced a waxy liver and a waxy spleen. We also found tuberculosis present.

DR. MORSE.—It seems to me that in the light of the knowledge which we now possess of the different forms of white corpuscles, as shown by differential staining, it is too late to speak of the relative proportions of white and red corpuscles in making the diagnosis between leucocytosis and leukæmia. The number of red corpuscles depends on one factor and the number of white corpuscles on another. There is no necessary relation between them. They must, therefore, always be considered independently. Moreover, there is no absolute number of white corpuscles which can be regarded as fixing the line between leucocytosis and leukæmia. In leucocytosis the increase is largely in the number of polynuclear neutrophiles, although in infants it may be in the lymphocytes. In splenic-myelogenous leukæmia, on the other hand, while all forms are increased, the bulk of the increase is due to the presence of abnormal white corpuscles, the myelocytes. The presence of a small percentage of myelocytes, even as high as 5 per cent., does not justify the diagnosis of splenic-myelogenous leukæmia, however, as a small percentage of myelocytes is found in many severe blood conditions, especially in children. The percentage of hæmoglobin in this case is much lower relatively than that of the red corpuscles, a condition which is the opposite of that usually excepted for pernicious anæmia. The number of leucocytes is increased, which is not

the rule in pernicious anæmia. This might, however, be due to some other cause and would not rule out pernicious anæmia. The histological changes in the red corpuscles alone correspond to the blood type of pernicious anæmia, but, as already pointed out, the tendency of the blood in disease in infancy is to revert the fœtal type, and the presence of poikilocytosis and of nucleated cells in infancy is of much less diagnostic value than in adults. As already stated, the case can only be regarded, therefore, as one of severe secondary anæmia with leucocytosis.

MELANOSIS LENTICULARIS PROGRESSIVA.

BY T. M. ROTCH, M.D.,

Professor of Diseases of Children, Harvard University.

During my service at the Children's Hospital in January, 1898, two children came under my observation who were under the care of my colleague, Dr. H. L. Burrill, to whom I am indebted for the privilege of reporting them.

The disease has been called by a number of names by various authors. Thus Kaposi speaks of it as xeroderma pigmentosum. Vidal calls it dermatosis Kaposi. Neisser calls it liodermia cum melanosi et telangiectasia. Taylor calls it angioma pigmentosum et atrophicum, while Pick and White speak of it by the name of melanosis lenticularis progressiva, which has now been quite generally adopted.

I shall not attempt to describe in detail a disease which especially belongs to the province of the dermatologist, but shall only say that it is exceedingly rare since, when it was first described by Kaposi in 1870, only about seventy-five cases have ever been recorded up to 1897. Nearly all the cases began in the first or second year, and one has been reported as occurring at five months. The predominance of the lesions is in the exposed parts of the body and the lesions consist of freckle-like spots of pigment, followed by atrophic degeneration of the skin and telangiectases. These are the primary lesions and may be followed later by more serious lesions of the skin, such verrucous growths starting usually in the pigmented spots and, according to Hyde, of either an epithelomatous, sarcomatous, or angiomatic character. The lesions may be single or many; may be confined to the skin or develop in the viscera, and usually lead to fatal results in a few or many years.

The etiology of the disease is exceedingly obscure, but it seems to have a congenital predisposition, as shown by the

occurrence of several cases in one family. Most of the cases have been females.

According to Dr. J. C. White, of Boston, none of the cases which have been observed in this country have occurred in native American stock, if such a type exists.

I present photographs of the two cases which I have just referred to. They are sisters and are of healthy parentage. The child on the left side of the picture is seven years old. When she was three months old small freckles appeared on her face and arms, growing larger, and becoming soft and fleshy in some places and in others horny. The lesions have especially affected her face and hands. She has been operated on eight times by curetting and skin-grafting, but the lesions have recurred in the scars.

A physical examination shows a fairly developed and nourished child with dark red hair. Scalp clean and clear. The disease is present on the face, neck, extensor surfaces of the arms and legs, in the clavicular region, and above the spines of the scapulæ. The lesions consist of brownish maculæ varying in size from a pin head to a split pea and are not elevated. There are numerous white horny growths, some with broad and some with constricted bases. On the face are several soft fleshy masses the size of a large pea, red and bleeding easily. Some of them are covered with a thick black crust. Between the various lesions the skin is atrophied in places, notably on the face and wrists. There is ectropion of both lower lids and there are no eyelashes. On the thighs and lower legs there are some very faint maculæ but no soft or horny growths. Nothing else abnormal is detected. The urine is normal. The blood shows

Leucocytes,	-	-	-	6,600
Erythrocytes,	-	-		4,400,000

A differential count shows

Neutrophiles,	-	-	-	72 per cent.
Small Mononuclears,		-	-	16 "
Large "		-	-	9 "
Eosinophiles,	-	-	-	3 "

The child has been treated with the mixed toxins of streptococcus erysipelatis and bacillus prodigiosus, one-fourth of a drop being given subcutaneously and increased daily. When



Fig. 1. MELANOSIS LENTICULARIS PROGRESSIVA OCCURRING IN SISTERS.
(CASES I. AND II.)

one drop was reached a reaction took place, the temperature rose to 102° F. with headache, vomiting and photophobia. The dose was then increased by one drop nearly every day until thirteen drops were reached. The condition steadily improved. The soft growths became smaller and less moist.

The crusts fell off and several warty growths disappeared from the face, neck and backs of the hands. When the treatment was omitted two days a very rapid relapse took place and new soft and horny lesions appeared. The softer ones grew larger and bled very easily; large black crusts formed and the skin between the lesions became red. On resuming the treatment improvement was noted in twenty-four hours. No especial effect was noticed where the injections were given in the fore-arms. When the child was exposed to sunshine the lesions became much worse, and when, after a week's exposure, it was returned to a darkened room, improvement was noticed at once not to be ascribed to toxins. The dose was gradually increased to eighteen drops, but the child appeared to become habituated to the toxin and it became harder and harder to get a reaction and to control the lesions, an omission of one day in the treatment causing a marked relapse. The general condition and health of child seemed to be very little impaired by the disease.

Various other forms of treatment were tried without effect, such as the application of copper sulphate, silver nitrate, ithyol, salicylic acid and various ointments.

The other sister, seen on the right of the picture, is six years old. The lesions appeared when she was five months old. Her general health is good. She has had five operations, curetting, grafting and plastic.

Physical examination shows the hair to be red, and the description of the lesions is the same as in her sister. She has ectropion of both eyes, and one large, soft, reddish growth at the outer canthus of each eye. On her hands and face are a few small white horny growths, and there are many small faintly red maculæ on her face, neck and extremities. She seems to be in an earlier stage of the disease than is her sister.

Judging from the experience of those who have treated this disease, Dr. Burrill decided to have this child placed in a large box and to have the light admitted through panes of glass of various colors, red and green. The child was exposed to the red rays from January 24th to March 4th. During this time the fleshy masses at the corner of the eyes became much smaller and dried up to a thin white crust, but no general improvement was noticed. From March 4th to April 5th green glass was substituted for the red, and no improvement was noticed. On

April 6th the combined toxins were again begun with no good results.

An official report by Dr. F. B. Mallory on a specimen of tissue from one of the facial tumors showed typical epidermoid cancer.



Fig. 2. MELANOSIS LENTICULARIS PROGRESSIVA.

(CASE I.)

In this case the toxin caused repeatedly attacks of redness, pain and swelling on the surfaces of the joints, and was finally omitted.

The result of the treatment of these cases shows that it was of no permanent benefit, and, in fact, there is no known curative

treatment for the disease. According to Hyde, most of the patients succumb to marasmus in from ten to twenty years.

In connection with these cases, Prof. James C. White, of Harvard, has sent me a report of a case which has been under his care, and which he has reported at length in the *Journal of Cutaneous and Venereal Diseases*, Vol. iii., December, 1885.

The case illustrates the essential and primary lesions of the disease melanosis lenticularis progressiva, namely, the pigmented appearance of the skin (melanosis), the atrophy or leucodermic condition, and the telangiectasis.

The case is interesting as one of the few which have reached adult life, the reason appearing to be that only the primary and benignant lesions of the disease developed in this case, and that the later lesions and verrucous growths which I have just spoken of did not occur. Where these later lesions do occur, especially if early in life, the disease is, as a rule, fatal.

SARCOMA OF THE KIDNEY IN AN INFANT NINE MONTHS OLD.

BY FRANK SPOONER CHURCHILL, M.D.,

Associate in Diseases of Children, Rush Medical College, in Affiliation with the University of Chicago; Professor of Pediatrics, Chicago Polyclinic.

I have to report the following interesting case: Otto W., male; nine months old, was brought to me at the Chicago Polyclinic, March 28, 1898, with the following history:

FAMILY HISTORY.—Paternal grandfather died from alcoholic excesses, whether from cardiac, hepatic, or renal trouble I could not learn. Father is a hard drinker; mother has drunk daily since birth, two glasses of beer, "to make milk for the baby."

PREVIOUS HISTORY.—Baby was breast-fed exclusively till three months; since then breast, cow's milk more or less diluted, bread, potato. Apparently well up to third month, when the present trouble began.

PRESENT ILLNESS.—About the third month of life, the mother noticed that the abdomen, on the left side, was beginning to swell, the increase in size being constant since then, and especially rapid the last few weeks; loss of flesh, nurses and eats but little; some vomiting, constipation, fretful, sleeps poorly.

PHYSICAL EXAMINATION.—Marked pallor and emaciation; weight, sixteen pounds five ounces; skin dry and harsh; distension of superficial veins of abdominal wall.

Chest.—Few coarse moist râles here and there; otherwise negative.

Abdomen.—Marked distension and prominence of whole of left side of abdomen, extending from left hypochondiac region diagonally down and to the right iliac region. Palpation over this swelling shows mass of comparatively regular outline, firm and hard, yet in places giving a sense of fluctuation, apparently tender to the touch, flat on percussion, not moving with respiration. No notch detected; no increase upward in area of splenic dulness; no increase in area of hepatic dulness.

Slight enlargement of glands in left inguinal region.

The blood and urine, examined two days later, showed following conditions: **BLOOD.**—Red, 3,104,000; white, 11,104; hæmoglobin, 25 per cent.; no plasmodium detected.

URINE (caught by mother in cup).—Smoky; much albumin (percentage not estimated); not enough for specific gravity.

Sediment.—One or two normal renal cells, considerable normal blood; many cells, large round and small round showing fatty degeneration. One or two compound granule cells; one quite extensive patch of round-celled epithelium (renal?); few leucocytes. One or two casts of urates, one or two fine granular casts of narrow diameter. Considerable fibrous débris.

DIAGNOSIS.—Sarcoma of left kidney.

While the position of the tumor of course suggested the possibility of its being splenic, the blood count, showing only 11,000 white corpuscles, ruled out all affections in which we might have had an enormously enlarged spleen with the history given. The tumor was evidently renal, and the age, history, and general condition all pointed to sarcoma. The examination of the urine showed apparently, extensive involvement of the kidney tissue itself. Among the round cells seen in the urinary sediment were some which did not have the exact appearance of renal epithelium, and I was inclined, somewhat rashly perhaps, to think that we had to do with a round-celled sarcoma, undergoing fatty degeneration, a clinical theory which subsequent pathological examination failed to confirm.

PROGNOSIS.—The prognosis was of course absolutely unfavorable, with or without operation. Dr. M. L. Harris, who kindly saw the case with me, advised against operation, but yielding to the urgent solicitation of the parents who were evidently anxious to cut short the child's misery, finally operated.

I quote from his detailed account of the operation:

“An incision below the twelfth rib extended transversely from a little to the left of the spinous processes to the linea semi-lunaris, and from thence upwards and downwards about 10 cms. The peritoneal cavity was opened. The descending colon had been carried anteriorly and towards the mid line and crossed the anterior surface of the tumor from above downwards. The tumor filled the concavity of the diaphragm above, extended to the pelvis below and considerably to the right of the mid line.

“The descending colon with its meso-colon were deflected inwards and the tumor enucleated.

“The spermatic and peri-renal veins were very much enlarged and had to be clamped at the lower pole of the tumor.

The renal vein with its branches spread out over the anterior surface of the tumor, the renal vessels were clamped and the tumor removed, and all hemorrhage effectually controlled. The immense wound was closed without drainage. Much to the surprise of every one the child stood the operation, which lasted about thirty minutes, remarkably well. The apparently good condition, however, did not last; death occurring a few hours later.

"The right kidney was not diseased; no metastatic growths were detected in the abnormal cavity."

Appended is the pathological report of the tumor carefully prepared for me by Dr. Maximilian Herzog, Professor of Pathology at Chicago Polyclinic:

"The tumor after removal weighed over three pounds; it is egg-shape in general outlines with diameters of $15\frac{1}{2}$, $10\frac{1}{2}$ cms., and possesses a fibrous capsule. On one side of the largest equator of the new growth there is situated what is left of the kidney, a flat mass more or less circular in outline, about 5 cms. in diameter and only a few mm. thick. When the tumor is laid open in its largest medium plane, it is seen to be divided into numerous compartments or cystic spaces—honey-combed.

"The compartments appear to contain blood, colloid and myxoid material. In the central axis of the tumor there runs a mass of tissue, several cms. long and about 1 cm. thick. This tissue macroscopically looks as if composed of striated muscle fibres. This impression, however, is erroneous as shown later by the microscopic examination.

"The whole neoplasm was fixed and hardened in Mueller's fluid (10 pts.) and Formalin (1 pt.), pieces from several places were subsequently dehydrated, embedded in paraffin, etc."

THE MICROSCOPIC EXAMINATION shows that the tumor is made up of a variety of heterogeneous elements; in some places where the neoplasm appears most dense the tissue is formed of densely packed spindle cells of a small type, with comparatively large spindle shaped or elongated oval nuclei, rich in chromatin of a very finely granular uniform type. These spindle cells surround small irregular blood-clefts or spaces. The latter have no walls proper, their boundaries being directly formed by the tumor cells. In other places we find the same type of small spindle cells not so densely packed, surrounding distinct vessels

(Fig. 1). Here it is evident that the tumor cells have been formed by a proliferation of the cells of the blood-vessel wall. Again in other places we find thin but distinctly walled blood-vessels surrounded by small round cells (Fig. 2). These round cells possess spherical vesicular nuclei fairly rich in chromatin of a coarse granular type and a very scanty amount of cell protoplasm. Free blood is found between the round cells just described.

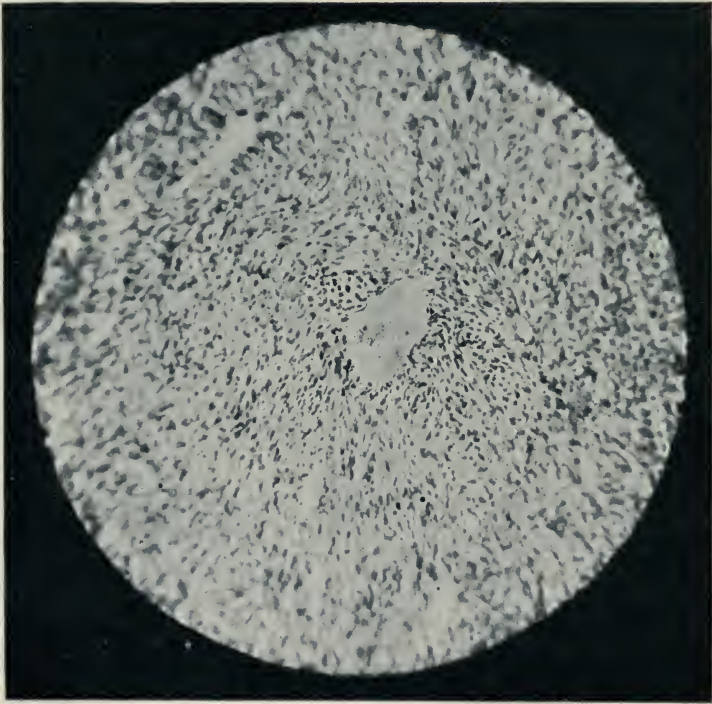


Fig. 1. MICROSCOPICAL SECTION OF KIDNEY TUMOR SHOWING TISSUE FORMED OF CLOSELY PACKED SMALL SPINDLE CELLS, SURROUNDING DISTINCT VESSELS.

The honey-combed spaces contain, as appeared on macroscopic inspection pseudo-mucinous and colloid material. Some of the cystic spaces represent large venous sinuses filled with blood.

What is left of the parenchyma of the kidney shows the evidences of very advanced atrophic processes, the uriniferous tubules being very small, collapsed, or compressed, and the

glomeruli small. Hyaline material is found in very few places, only in small blood-vessels and tubules. But outside of this there are found no evidences of degenerative processes of the renal parenchyma either in tubular epithelium or the glomerular capillaries. The only interstitial change found in that part of the kidney appearing as such on naked eye examination, is thickening of the capsule overlying the thin atrophic renal parenchyma.

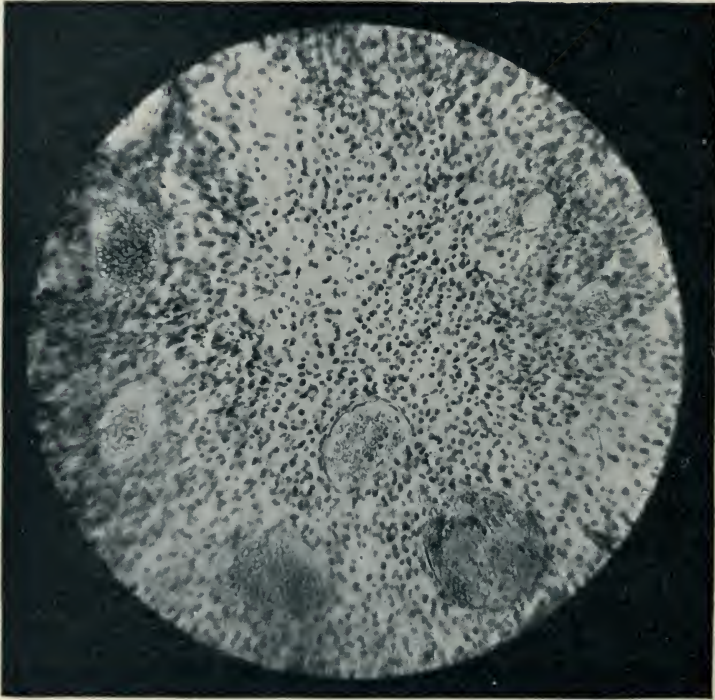


Fig. 2. MICROSCOPICAL SECTION OF KIDNEY TUMOR SHOWING THIN BUT DISTINCTLY WALLED BLOOD-VESSELS SURROUNDED BY SMALL ROUND CELLS.

Remnants of the organ were recognized on macroscopic inspection, while microscopic examination showed parts of the parenchyma in several places in the capsule of the tumor.

The central mass, which looked as if composed of muscle fibres, showed a very small number of nucleated round and spindle cells. The great majority of the cells found in this tissue have completely degenerated, their nuclei have disap-

peared and their cell protoplasm is granular and cloudy. The cell boundaries are frequently indistinct. These degenerated cells are imbedded in a stroma of a fibrinous material, the fibres of which are arranged in more or less distinctly parallel bundles and groups. To this arrangement is due the naked eye impression that this tissue contained striated muscle cells.

What appeared macroscopically as the capsule of the tumor is shown to be made up externally of perfectly hyaline non-nucleated fibres. Internally we have a tissue consisting of slender nucleated connective tissue spindle cells in which there are found here and there some few very small and atrophic uriniferous tubules, also some almost unrecognizable glomeruli.

From the above description it appears that the neoplasm is an angio sarcoma of a mixed cell type with advanced secondary degenerative changes. The neoplasm evidently started from the vessels.

It also appears that the tumor took its origin somewhere in the pelvis of the kidney, and in growing, compressed the parenchyma and caused it to undergo "pressure atrophy."

The chief interest of these malignant tumors of the kidney in children of course centres in their etiology. The view generally accepted at present is that of Cohnheim, that they are of congenital origin and due to misplaced embryonic tissue. He supposes that in the development of the kidney embryonic cells from the surrounding structures are incorporated in its capsule and subsequently give rise to a new growth.

The Wolffian body comes from that part of the nephrotome, which is near the myotome; thus it is easy to see that some of the mesothelial cells may become incorporated in the capsule and subsequently develop into striped muscle tissue strikingly like embryonic muscle. Such tissue has been observed in some cases reported in literature; none were found in the specimen from this case.

An exhaustive review of this subject has been published by Wentworth,* and by Walker.† Since this paper was written an abstracted account of a successful operation by J. C. Cotton for renal sarcoma in a nine-months' infant has been published in the current (June) number of the ARCHIVES OF PEDIATRICS. The

* Archives Pediatrics—Vol. xiii., No. 5.

† Annals of Surgery—Vol. xxvi., p. 529.

infant lived nearly one and a half years after the operation, and finally died of a recurrence of the disease. The autopsy showed three tumors, all originating in the lumbar glands.

583 EAST DIVISION STREET.

DISCUSSION.

DR. MORSE.—I would differ a little from Dr. Churchill's conclusions on the examination of the blood. He said, I think, that the examination of the blood justified ruling out enlargement of the spleen. That does not seem to me to be quite right, as it is perfectly possible to have an enlargement of the spleen with the usual number of leucocytes.

DR. JENNINGS.—A case came under my observation during the past year in which a tentative diagnosis of sarcoma of the kidney was made, in which it was impossible to find evidence by the presence of leucocytosis of splenic enlargement and it was impossible to find the notch of the spleen. The case proved, however, to be one of splenic enlargement. An interesting fact perhaps was the absence of any urine changes in this case, which made the diagnosis of a renal tumor very problematic.

DR. JACOBI.—The case is of interest for several reasons. First, because these tumors that contain striated muscular fibres are very rare. They have been included under the term rhabdomyomata. The first, or one of the first cases, was published some twenty years ago by Brozin, a Swiss. Since that time, up to 1884, when I presented my paper on this subject before the Copenhagen Congress, five or six such rhabdomyomata have been observed. The cases at that time I could collect of sarcoma of the kidneys, including the number I had seen, was forty odd. That, I might state, was the first time that a differential diagnosis between sarcoma and carcinoma of the kidney was made. Formerly they went either under malignant tumors of the kidney or under the head of carcinoma. There is one diagnostic point of importance that might have misled the doctor, but did not. That is, in sarcoma the urine in 80 per cent. of the cases does not contain any blood or any kidney elements. There is no nephritis with it. Most of the tumors are of the exact nature of this. They are not tumors of the kidneys, but tumors outside the kidneys, lying between the capsule and the kidneys and compressing the latter. There is no nephritis and no blood. There is blood in a large percentage of cases of carcinoma and the presence of blood is more in favor of carcinoma than of sarcoma. Another point of interest in this case was that the child died so

early. Most of them are not so badly off when they are nine months old, but many of them live two, or three, or four years. I have seen a number of them live to be five years of age. I collected, in 1884, cases that had lived nine, eleven and thirteen years. Usually sarcoma of the kidney is only unilateral. Metastases do not take place until very late, sometimes not at all. Generally the abdominal lymph bodies are not affected. That is why these cases can live so long.

DR. FRUITNIGHT.—I would like to call to the memory of the Society a case of sarcoma of the kidney which I presented at the meeting of the Virginia Hot Springs in which there were striped fibres, showing that it was a rhabdomyo-sarcoma.

TWO CASES OF PROLONGED PNEUMONIA.

BY FLOYD M. CRANDALL, M.D.,

Adjunct Professor of Pediatrics, New York Polyclinic; Visiting Physician,
Hospital for Scarlet Fever and Diphtheria; Consulting Physician,
Children's and Infants' Hospitals, New York.

The two following cases are reported as being illustrative of the prolonged course which broncho-pneumonia sometimes runs. The first is quite typical of broncho-pneumonia complicating pertussis. The second is anomalous and typical of no ordinary type.

The first patient was a boy of twelve months, who had been ill three days at the time of my first visit. The onset of the acute symptoms had been somewhat sudden and occurred during the fourth week of whooping-cough. When first seen the child exhibited the familiar symptoms of pneumonia. Pulmonary consolidation could not be detected, but in the left lower lobe posteriorly, fine crepitating râles were heard over a considerable area and there was slight dulness on percussion. Actual consolidation was not detected until the sixth day. The case pursued a slow but typical course; the temperature began to fall on the fifteenth day and on the nineteenth day the child was markedly improved in all respects. Sudden rise of temperature on the following day, however, was followed by evidence of extension of the pneumonic process. An elevation of temperature on the twenty-sixth and twenty-seventh days was accompanied by new but rather indefinite physical signs on the other side of the chest. Following this the child gradually began to improve and seemed decidedly better. The consolidation, however, remained but little changed and the temperature never returned entirely to normal. On the thirty-sixth day the child again became worse. The temperature slowly arose from about 100° to 103° ; evidences of consolidation appeared in the right lower lobe, and the child died on the forty-third day of the pneumonia. During the first four weeks the whooping-cough was extremely severe, the paroxysms being frequent and distressing. They gradually subsided, however, during the fifth week and were not severe during the last days of the child's life. The

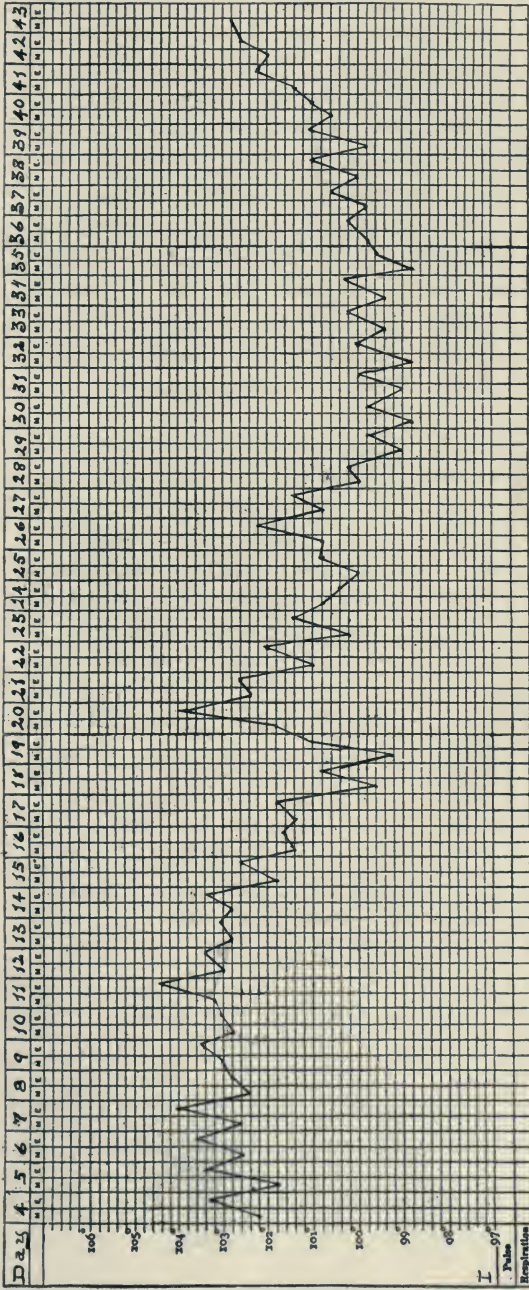


Chart I. TEMPERATURE RANGE IN A CASE OF PROLONGED PNEUMONIA, COMPLICATING WHOOPING-COUGH AND TERMINATING FATALLY.

child died apparently from exhaustion and not from the acuteness of the pneumonic symptoms.

The accompanying temperature chart shows four distinct febrile periods. Each of these was accompanied or soon followed by the evidences of fresh pneumonic consolidation.

There is nothing unusual in this history or temperature chart, except that the duration of the illness was somewhat longer than commonly occurs even in prolonged pneumonia. Cases of longer duration have been reported to this society and have shown, also, the same peculiar periods of increased fever. A prolonged and tedious course is quite characteristic of pneumonia when it complicates whooping-cough. In my experience it has been a particularly trying and fatal disease.

The second case was an extraordinary one, owing to the duration of the disease, the remarkable manner in which the child tolerated the fever, and the peculiar complication which seemed to have some active part in prolonging its course.

On December 16, 1895, I was summoned in haste to see A. D., a girl of twenty months. While playing with popcorn, some of which she was then eating, she suddenly choked. She coughed violently, and became so cyanotic that the mother was alarmed and sent for me. She was relieved after a few minutes, however, and when I reached her was apparently perfectly well except for a slight, loose cough which she had had for several days. There was no temperature nor dyspnoea. The mother was quite sure that some of the corn had been drawn into the larynx and had not been coughed out. A careful examination of the chest, however, revealed nothing except a moderate number of moist râles upon both sides posteriorly. Two days later I was again summoned and found that the child had been ill through the night. The cough had increased, fever had developed, and the child was restless and irritable. There was nothing, however, to indicate more than bronchitis. The temperature was 101° and rose to 102° in the evening.

On the following morning the temperature was 104.5° and the child presented the appearance so characteristic of pneumonia. The chest on both sides was filled with small râles. The physical signs, however, were not yet positive. Without going into further details, it is sufficient to say that the child passed through a very characteristic and somewhat prolonged

attack of broncho-pneumonia. Consolidation was detected clearly on the fourth day. The first evidences appearing in the upper portion of the right lower lobe posteriorly. The progress of the disease was rather slow, the temperature with one or two exceptions not going below 100° until the seventeenth day, and not actually reaching the normal until the twenty-third day. On the eighteenth day the temperature rose to 104° but fell immediately after the action of the cathartic. It was clearly due to indigestion.

The consolidation became quite extensive, involving all the middle portions of the right lung. It was slow in resolving and a bronchial element could be detected in the respiration on the twenty-ninth day, although the temperature had been practically normal for more than a week.

On the thirtieth day the temperature rose to $100.2-5^{\circ}$; the child began to sneeze and cough; the eyes became watery and red; and all the classical symptoms of grippe appeared. That disease was then prevailing extensively. Two older children in the family were already suffering from grippe, and the trained nurse became ill with it on the preceding day.

In view of the child's condition and the signs still present in the chest, I was very sorry to see an elevation of temperature and pursued a plan of treatment not common with me, that of administering antipyretic doses of phenacetine, together with small doses of salol. I did this with the hope of breaking the force of the grippe and preventing the recurrence of the pneumonia. The treatment was continued for twenty-four hours, but the temperature immediately arose when it was discontinued. The effect of this treatment is shown by the chart, where a fall of temperature will be observed on the thirty-first and thirty-second days. Signs of consolidation rapidly developed in the original seat of disease in the right chest. The consolidation slowly extended until it apparently involved the complete right side from the apex to the base posteriorly. In front there were small areas where the consolidation did not seem complete. The fever continued without remission for thirty-one days and the child presented all the classical signs and symptoms of broncho-pneumonia. On the twenty-sixth day of the second attack, I again attempted by the use of phenacetine to reduce the temperature, as the disease in the chest was apparently not extending, but upon its discontinuance after twenty-four hours the temperature ranged higher than before.

The condition of the child during the last two weeks of this period was very surprising. Although she had become wasted and emaciated to an extreme degree, she was much of the time quite bright and happy. It was impossible to keep her from sitting up in bed a part of the time, and she would play quietly for a half-hour at a time with toys or picture books. She was learning to talk, and learned many new words during the illness. She was seen several times by Dr. Holt, both during the first and the second attack. At his last two visits, upon seeing the temperature chart before seeing the child, he expressed a most unfavorable opinion, upon the general principle that such prolonged cases usually terminated fatally from exhaustion of the child. Upon seeing the patient, however, his prognosis was favorably modified.

During the whole second attack there was a paroxysmal and very distressing cough, causing the child at times to vomit; for a few days nourishment was almost entirely rejected. It was not, however, whooping-cough. As confirmatory of this belief is the fact that neither of the other children in the family had any cough. This cough became more paroxysmal and more difficult, and was evidently beginning to sap the child's strength. For three days it was particularly troublesome, and the temperature ranged higher than it had done for many days.

On the thirty-first day of the second attack and the sixtieth of the illness, after a prolonged and extremely violent attack of coughing, a very large mass of thick green mucus, having a bad odor, was expectorated. The nurse was about to throw it away when something attracted her attention. She found a very tough greenish mass of mucus in the centre, in which was imbedded a dark, irregular object. She washed and picked the mucus away, and saved the central substance. When I saw it, it had, she said, shrunken in size as it dried, and it is now smaller than it was then. It is now light and spongy, and appears like worm-eaten cork. It seems entirely easy to believe that it is the central hard portion of a piece of popcorn. Its long maceration had so changed it, however, that it is impossible to say that this is the fact either from its gross or microscopical appearance.

At Dr. Holt's first visit, he was told of the occurrence of the coughing attack preceding the onset of the disease, but by physical examination, did not find anything to indicate a foreign body in the bronchi.

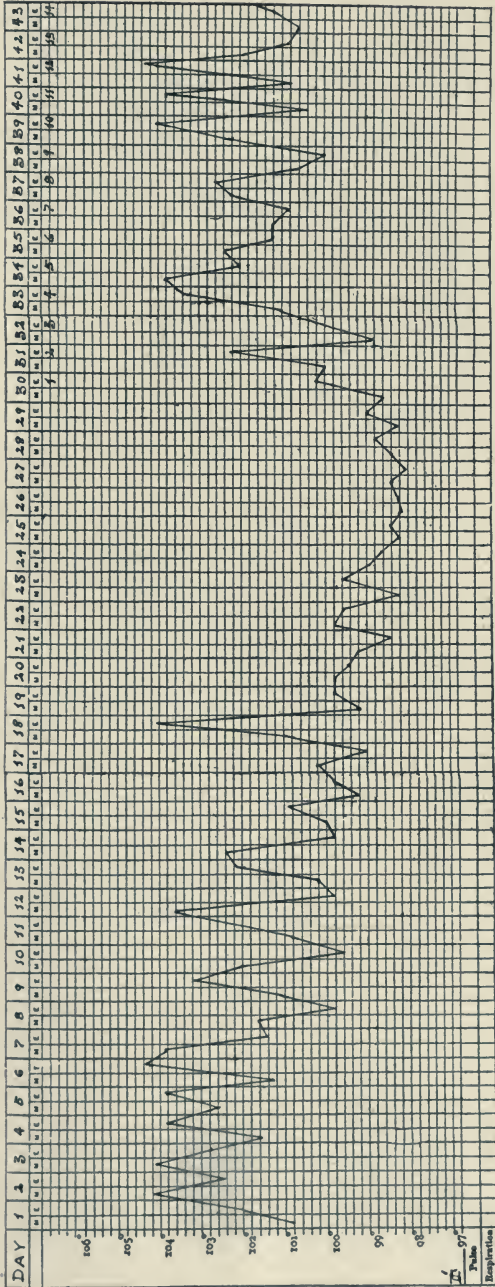


Chart II. TEMPERATURE RANGE IN A COMPLICATED AND PROLONGED CASE OF PNEUMONIA, TERMINATING IN RECOVERY.

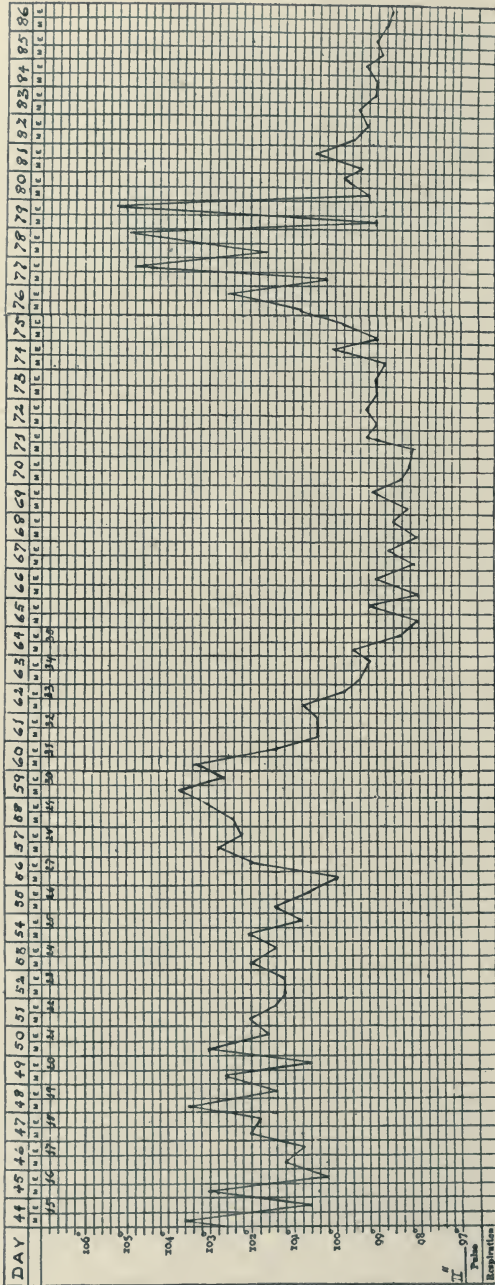


Chart II. TEMPERATURE RANGE OF PREVIOUS PAGE (CONTINUED).

This foreign body was expelled during the forenoon; the temperature that morning had been over 103° , at which point it had been ranging for three days. That afternoon the temperature fell to nearly 101° . It steadily declined during the next three days, and reached normal. The child's general condition at once began to improve. Evidences of resolution were soon recognized in the chest, but complete resolution was very slow. Bronchial respiration and dulness on percussion were heard in the central portions of the chest, where the consolidation first appeared, for six or seven weeks, and had not entirely disappeared six months later. At the end of a year, there was still dulness on percussion over the right side of the chest, but respiratory sounds were quite normal through its whole extent. During the following winter the child developed rapidly, mentally and physically, and she appeared like a perfectly healthy and well child.

A still further complication remains to be described. Ten days after the temperature had reached normal, when everything seemed to be going well, the temperature again began to rise, there being a distinct remission in the forenoon. At the same time the other members of the family began to have similar symptoms, which were clearly malarial in their origin. Preceding this the street in front of the house had been dug up for the repair of a sewer. A break in the house pipe was also found in the cellar. The character of this attack was not at first detected, as the signs of consolidation became more marked with the appearance of the fever. It was feared that the pneumonia was again recurring, although the cough and other symptoms had not increased. After full doses of quinine, the temperature returned rapidly to normal, and did not at any time again rise above 100° .

The part played by the foreign body expectorated on the sixtieth day is, to my mind, not entirely clear. The attack of strangling two days before the onset of pneumonic symptoms with almost continuous illness for nine weeks, the peculiar paroxysmal cough which rapidly disappeared, the fall of temperature which began sharply on the day the foreign body was ejected, certainly lead one to suspect that it had an influence in prolonging the course of the disease.

A REPORT ON CASES OF MEASLES.

BY WALTER LESTER CARR, M.D.,

New York.

The patients who furnish the subject of this report were seen in the Infants' and Children's Hospitals, Randall's Island, during a service of two months, namely from February the first to March the thirty-first of this year. The total number of cases under observation was 115. There were 107 cases of rubeola and 8 cases of rubella. The number of uncomplicated cases of rubeola was 44; rubella, 6; of complicated rubeola, 62; of rubella, 3.

There were 65 boys and 50 girls. The oldest child, a boy, was sixteen years; the youngest, a boy, was five months. The average age over two years was 71 11-15 months. The average age under two years was 14 19-26 months. The average age of all cases was 45 5-14 months.

The average duration of uncomplicated cases from the prodromal symptoms to the end of desquamation was twenty-six days.

Sixty-two of the patients with rubeola had complicating diseases and 45 had the usual laryngeal and bronchial catarrh.

COMPLICATIONS.

Rubeola, broncho-pneumonia,	9
“ “ “ enteritis,	3
“ “ “ gastro-enteritis,	1
“ “ “ pertussis,	2
“ “ “ pleurisy with effusion, acute diffuse nephritis,	1
“ “ “ pertussis, enteritis,	1
“ “ “ acute miliary tuberculosis,	1
“ “ “ general tuberculosis,	1
“ “ “ purulent synovitis of hip,	1
“ “ “ enteritis, pleurisy, effusion,	1

Rubeola, pertussis,	2
“ “	otitis, media suppur.,	3
“ “	corneal ulcer,	1
“ “	lobar pneumonia,	1
“ “	scarlet fever,	1
“ “	lobar pneumonia, varicella,	1
“	bronchitis,	3
“ “	diphtheria,	1
“ “	otitis media suppur.,	1
“ “	“ “ “ enlarged submaxillary gland,	1
“ “	abscess popliteal space,	1
“ “	laryngitis,	2
“	otitis media suppur.,	3
“ “	“ “ “ enteritis,	1
“ “	“ “ “ tonsillitis,	1
“	enteritis,	5
“	entero-colitis,	1
“	gastro-enteritis,	1
“	lobar pneumonia,	1
“ “	“ “ pleurisy, effusion,	1
“ “	“ “ membranous laryngitis,	1
“	general tuberculosis,	1
“	nasal diphtheria (K.L.B. present),	2
“	scarlet fever,	1
“	varicella, membranous tonsillitis (clinical diphtheria, no K.L.B.),	1
“	marked conjunctivitis,	1
“	membranous tonsillitis (no K.L.B.),	1
“	catarrhal laryngitis,	1
	Total,	62

The prodromal period of the measles that developed in well children did not present any unusual severity and not one of the patients had convulsions. Children who were ill with other diseases, (*i.e.*, pertussis, enteritis,) had more severe initial catarrh but no higher fever than the children who were in good health at the onset of the disease.

The eruption was observed in every case where the throat was examined and was noted as present on the soft and hard palate twenty-four to forty-eight hours before the appearance of the exanthem.

When first questioned as to Koplik's symptom the members of the house staff did not note its presence and I was rather inclined to doubt that it could be always found. After a study of some of the later cases of the epidemic and a comparison of the buccal eruption of children seen in private practice, I am convinced that the "bluish-white speck in the centre of a

reddish spot" is to be regarded as a valuable sign of beginning measles.

The average duration of the uncomplicated cases from the first prodromal symptoms to the end of desquamation was twenty-six days.

The average duration of the eruption from the first appearance on the face to its last staining was noted in forty-nine cases and averaged 7.56 days.

The average duration was increased by two or three cases. In one, a baby of eleven months, a bottle-fed infant who was poorly nourished, the eruption lasted seventeen days.

A baby of eighteen months, who was ill with measles, broncho-pneumonia, pleurisy with effusion and enteritis, had an eruption that left its staining for nineteen days.

The cases where the eruption was persistent were usually those of pneumonia or a low vitality. On the other hand the eruption was recorded as of short duration in babies with broncho-pneumonia.

There was not a case with hemorrhagic measles and only one child showed any tendency to a purpuric eruption, and as this child was suffering with a suppurating hip, it may be questioned how much of the character of the eruption was due to the measles and how much to the joint disease.

Rubeola was recorded as having attacked four children four weeks after the first eruption. The eruption in the first attack had shown itself from five to seven days in each case and the eruption at the second appearance lasted from five to thirteen days. The child on whom the exanthem was slow in fading did not have any complication. The eruption, course of the disease, character and general symptomology were identical with the first attack of the measles.

As the rubeola was so prevalent in the hospitals and schools a number of children were exposed to a reinfection and it was supposed that the second attack of measles developed from such exposure.

The eruption in the rubella cases was more evanescent than in the cases where measles was diagnosed. There was in these children a slight redness of the fauces and the enanthem lasted but a short time. It was impossible to determine, with accuracy, the enlarged lymph nodes as a large proportion of the measles cases had the enlargement, and children who were not

ill were frequently suffering from the effects of catarrh and engorgement of the nodes. The rubella eruption was not noted as crescentic in its distribution and it was an irregularly diffused macular eruption with erythematous patches on the legs and arms. It gave little trouble and no constitutional symptoms or complications followed.* The temperature was low and did not go over $101\frac{1}{2}^{\circ}$, to 102° F.

Five children who had rubeola had afterward a rubella. In most of these latter cases the eruption was evanescent, the symptoms were slight and the systemic disturbance was not marked by any complication. One child with a severe enteritis showed a persistence of the cutaneous eruption for over a week. There was no desquamation detected.

Twelve cases were transferred to the Health Department Hospital on North Brother Island. It is interesting to record that the cases sent to the Health Department were serious and almost all of them were complicated.

3	Measles and diphtheria,	3	recovered
2	" "	pertussis,	.	.	.	1	died. 1 recovered
1	" "	pertussis and broncho-pneumonia,	.	.	.	1	died
1	" "	scarlet fever,	.	.	.		1 recovered
1	" "	scarlet fever, broncho-pneumonia,	.	.	.	1	died
1	" "	broncho-pneumonia, enteritis,	.	.	.	1	died
1	" "	broncho-pneumonia,	.	.	.	1	died
1	" "	marasmus,	.	.	.	1	died
1	" "	uncomplicated	.	.	.		1 recovered
		Total,	.	.	.	6	died 6 recovered

The complication that existed in four of the six fatal cases was broncho-pneumonia. One child, who died, after measles had pertussis, broncho-pneumonia and stomatitis gangrenosa.

Fourteen infants and children died while in the hospital and the diseases of which they died may be classified as follows:

Broncho-pneumonia,	suppurating hip,	1
" "	suppurating pleurisy,	1
" "	tuberculosis, meningitis, ileo-colitis,	1
" "	miliary tuberculosis, enteritis,	1
" "	rhachitis,	1
" "	chronic tuberculosis,	1
" "	pleurisy, sero-purulent, acute diffuse nephritis,	1
" "	gastro-enteritis, rhachitis,	1

* One child of six years had lobar pneumonia three weeks after the rubella. The pneumonia was ushered in with a chill and high temperature. It terminated by crisis on the seventh day. The pneumonia was limited to the middle of the left lung.

TABLE SHOWING THE COURSE OF THE DISEASE IN 107 CASES OF RUBEOLA.

NAME	AGE	SEX	DIET	Month and Day of Invasion	Appearance of Rash	Disappearance of Rash	COMPLICATIONS	GENERAL CONDITION	RESULT
S. P.	16 mos.	F.	Regular	3-24	3-29	3-30	Broncho-pneumonia.	Fair. Well developed.	Died.
A. A.	19 mos.	M.	Regular	3-7	3-10	3-19	Bron.-pneu. Enteritis.	Poorly nourished. Rachitis.	Recovered.
M. L.	10 mos.	F.	Breast	3-24	3-25	3-31	None.	Very good. Well developed.	Recovered.
C. H.	12 mos.	M.	Breast	3-12	3-13	3-20	None.	Poor. Hydrocephalic.	Recovered.
R. M.	9 mos.	F.	Wet Nurse	3-14	3-15	3-20	Gastro-enteritis.	Poor. Poorly developed.	Died.
J. F.	12 mos.	M.	Regular	3-9	3-11	3-15	Tuberculosis (?)	Poor. Poorly developed.	Recovered.
E. L.	9 mos.	F.	Wet Nurse	3-18	3-19	3-26	Broncho-pneumonia.	Poor. Markedly rachitic.	Recovered.
K. G.	11 mos.	F.	Bottle	3-9	3-10	3-27	2d rash (rubella).	Poor. Poorly developed.	Died.
E. W.	20 mos.	F.	Regular	3-31	4-3	4-10	None.	Good. Well developed.	Recovered.
C. S.	8 mos.	F.	Bottle	2-19	2-21	—	None.	Marked rachitis (negro).	Recovered.
N. B.	23 mos.	M.	Bottle	3-4	3-6	—	None.	Very poor. Chr. meningitis.	Recovered.
F. S.	12 mos.	M.	Bottle	3-2	3-4	3-12	Enteritis, Otitis Med. Sup.	Very poor. Poorly developed.	Recovered.
B. G.	24 mos.	F.	Regular	3-8	3-9	3-14	Bronchitis.	Good. Well developed.	Recovered.
L. S.	23 mos.	M.	Regular	3-22	3-24	—	K. L. B. in nose.	Good. Well developed.	Sent to N. B. I.
R. R.	13 mos.	M.	Bottle	3-8	3-11	—	Broncho-pneumonia.	Poor. Flabby. Anaxmic.	Recovered.
P. B.	7 mos.	M.	Wet Nurse	2-26	2-28	3-18	Bron-pneu. 2d rash (rubella), 3-12	Poorly developed. Rachitis.	Died.
E. V.	19 mos.	F.	Regular	3-23	3-25	—	Bron-pneu. gastro-enteritis.	Poor. Rachitic.	Died.
J. E.	11 mos.	M.	Breast	3-27	3-30	—	Bron-pneu. (intense congestion).	Poor. Fair development.	Recovered.
J. G.	5 mos.	M.	Regular	3-7	3-9	3-16	Pertussis. Corneal ulcer.	Good. Well developed.	Recovered.
W. S.	3½ yrs.	M.	Regular	3-5	3-9	3-14	Pertussis.	Good. Well developed.	Recovered.
J. S.	4 yrs.	M.	Regular	3-6	3-8	3-14	Pertussis, Otitis Med. Sup.	Poor. Well developed.	Recovered.
J. L.	2½ yrs.	M.	Regular	3-13	3-15	3-20	Pertussis, Lobar pneu.	Fair. Well developed.	Recovered.
H. T.	24 mos.	M.	Regular	3-8	3-9	3-11	Pertussis, Otitis Med. Sup.	Poor. Tuberc. rt. hip.	Recovered.
F. de N.	20 mos.	M.	Regular	3-15	3-19	3-24	Lobar pneumonia.	Fair. Marked rachitis.	Died.
B. C.	2½ yrs.	F.	Regular	2-23	2-28	3-7	Pertussis, Bron-pneu.	Fair. Well developed.	Died.
C. A.	2½ yrs.	M.	Regular	2-13	2-14	2-22	Pertussis, Lobar pneu. Varicella.	Poor. Well developed.	Sent to N. B. I.
I. L.	3½ yrs.	F.	Regular	3-2	3-5	—	Pertussis. Scarlet fever.	Good. Well developed.	Recovered.

TABLE SHOWING THE COURSE OF THE DISEASE IN 107 CASES OF RUBEOLA—Continued.

NAME	AGE	SEX	DIET	Month and Day of Invasion	Appearance of Rash	Disappearance of Rash	COMPLICATIONS	GENERAL CONDITION	RESULT
M. S.	27 mos.	F.	Regular	3-29	3-31	—	{ Pertussis, Broncho-pneumonia. Noma.	Good.	Sent to N. B. I. Died.
F. F.	22 mos.	M.	Regular	3-31	4-1	—	None.	Fair.	Recovered
M. C.	21 mos.	F.	Regular	3-30	4-1	—	Broncho-pneumonia, Pertussis.	Fair.	Sent to N. B. I.
E. L.	8 mos.	M.	Breast	3-22	3-23	—	Pertussis, Otitis Media.	Good.	Recovered.
I. C.	22 mos.	F.	Regular	3-9	3-11	3-18	Bronchitis, Diphtheria.	Good.	Sent to N. B. I.
S. T.	1 yr.	M	Regular	—	—	—	Scarlet-fever, Broncho-pneu.	Fair.	Recovered.
L. S.	16 mos.	F.	Regular	3-23	3-24	—	K. L. B. in nose.	Good.	Sent to N. B. I.
J. Q.	12 mos.	F.	Breast	2-24	2-27	3-6	Otitis Media.	Good.	Recovered.
H. N.	23 mos.	M.	Regular	3-22	3-26	4-2	{ Clin. Symp. of Diph., no K. L. B., Varicella, Pseudo-mem. on tonsils.	Good. Fair.	Recovered.
C. T.	25 mos.	F.	Regular	3-27	3-29	4-3	None.	Good.	Recovered.
F. H.	2 yrs.	M.	Regular	2-5	2-8	2-14	Bronchitis, Otitis Media.	Poor.	Recovered.
J. G.	10 mos.	F.	Bottle	2-10	2-14	2-20	Otitis Media.	Good.	Recovered.
M. Z.	7 yrs.	F.	Regular	2-8	2-10	2-20	Laryngitis, Bronchitis.	Good.	Recovered.
A. G.	7 yrs.	M.	Regular	2-11	2-13	2-19	Marked Conjunctivitis.	Good.	Recovered.
T. L.	7 mos.	M.	Bottle	3-20	3-22	—	None.	Poor.	Recovered.
C. P.	2½ yrs.	M.	Regular	2-15	2-17	3-1	Enteritis.	Very poor.	Recovered.
M. G.	4½ yrs.	F.	Regular	2-28	3-1	3-11	None.	Good.	Recovered.
J. B.	16 yrs.	M.	Regular	1-28	1-28	2-8	None.	Good.	Recovered.
M. G.	6 yrs.	M.	Regular	1-29	2-31	3-10	Bronchitis.	Good.	Recovered.
M. McC.	10 yrs.	F.	Regular	2-7	2-10	2-20	None.	Good.	Recovered.

Hereditary Syphilis.

Nystagmus.

TABLE SHOWING THE COURSE OF THE DISEASE IN 107 CASES OF RUBEOLA—Continued.

NAME	AGE	SEX	DIET	Month and Day of Invasion	Appearance of Rash	Disappearance of Rash	COMPLICATIONS	GENERAL CONDITION	RESULT
B. C.	8 yrs.	F.	Regular	2-2	2-3	?	None.	Good.	Recovered.
O. C.	10 mos.	F.	Breast	2-10	2-16	2-21	Broncho-Pneumonia, Enteritis.	Poor.	Died.
M. G.	12 yrs.	F.	Regular	2-3	2-6	2-13	None.	Good.	Recovered.
J. W.	4 yrs.	M.	Regular	2-10	2-12	2-18	None.	Good.	Recovered.
G. B.	16 mos.	M.	Regular	3-10	3-14	3-27	{ Bronchitis, Otitis Med. Sup., Enlarged Submaxillary Glands.	Fair.	Recovered.
J. M.	18 mos.	M.	Regular	3-10	3-12	3-19	{ Membranous Tonsillitis 4 days. Culture negative.	Fair.	Recovered.
H. B.	2 yrs.	M.	Regular	3-16	3-18	—	Lobar pneu., mem. laryngitis.	Poor.	Died.
M. K.	4 yrs.	F.	Regular	?	2-12	?	None.	Good.	Recovered.
G. W.	4 yrs. 9 mo	M.	Regular	?	2-7	?	None.	Good.	Recovered.
H. F.	2 yrs.	M.	Regular	2-23	2-23	2-29	None.	Good.	Recovered.
F. Mcl.	10 y. 6 mo	M.	Regular	1-28	2-28	3-7	None.	Fair.	Recovered.
W. W.	6 yrs.	M.	Regular	2-8	1-30	2-8	None. { 2d rash (rubella), 3-25. 2d rash disappeared, 3-29. Bronchitis.	Good.	Recovered.
M. S.	4 yr. 3 mo	M.	Regular	2-8	2-10	2-27	None.	Good.	Recovered.
M. L.	13 y. 6 mo	F.	Regular	2-2	2-3	?	None.	Good.	Recovered.
B. C.	14 yrs.	F.	Regular	?	?	?	None.	Good.	Recovered.
F. K.	8 yr. 9 mo	F.	Regular	?	?	?	None.	Good.	Recovered.
J. R.	?	F.	Regular	?	2-30	?	None.	Good.	Recovered.
L. C.	9 yrs.	F.	Regular	?	3-3	?	None.	Good.	Recovered.
E. B.	6 1/2 yrs.	F.	Regular	?	3-2	3-7	None.	Good.	Recovered.
M. S.	2 yr. 1 mo	F.	Regular	3-5	3-10	3-12	Bronchitis.	Good.	Recovered.
H. H.	23 mos.	M.	Regular	2-23	2-25	3-6	Broncho-pneumonia.	Good.	Recovered.
K. G.	2 yr. 9 mo	F.	Regular	3-3	3-10	3-16	Otitis Med. Sup. Tonsillitis.	Poor. Tuberculous.	Recovered.

NOTE.—Cases marked ? had their records kept but they were destroyed by mistake during fumigation of the hospital pavilions and the notes could not be completed.

TABLE SHOWING THE COURSE OF THE DISEASE IN 107 CASES OF RUBEOLA—Continued.

NAME	AGE	SEX	DIET	Month and Day of Invasion	Appearance of Rash	Disappearance of Rash	COMPLICATIONS	GENERAL CONDITION	RESULT
M. C. O'C	1 yr. 3 mo	F.	Regular	2-3	2-6	?	{ Bron.-pneumonia. Pleurisy. Eftusion. Acute diffuse nephritis. None. None.	Poor. Very Anæmic. Good. Good.	Died. Recovered. Recovered.
I. R.	9 yrs.	M.	Regular	2-16	2-18	?	{ Gastro-enteritis. Broncho-pneumonia. Pleurisy.	Poor.	Sent to N.B.I. Died.
M. B.	8 yr. 6 mo	M.	Regular	?	?				
J. T.	2 yr. 1 mo	M.	Regular	?	3-28	4-1	{ Broncho-pneumonia. Pertussis.	Good.	Sent to N.B.I. Died.
F. M.	1 yr.	M.	Breast	3-24	3-26	—			
W. M.	4 mos.	M.	Breast	3-16	3-17	3-30	{ Bron.-pneumonia. Enteritis.	Poor.	Sent to N.B.I. Died.
C. D.	2 yr. 2 mo	F.	Regular	3-16	3-25	3-31	{ Catarrhal laryngitis.	Good.	Recovered.
M. L.	1 yr. 6 mo	F.	Regular	2-1	2-3	?	{ None.	Good.	Recovered.
M. S.	1 yr. 2 mo	M.	Regular	2-22	2-26	3-1	{ None.	Good.	Recovered.
L. L.	6 mos.	F.	Bottle	2-14	2-17	2-25	{ Marasmus. Enteritis.	Amaturotic fam. Idiocy.	Recovered.
M. C.	8 mos.	F.	Breast	2-15	2-17	?	{ Broncho-pneumonia.	Fair.	Recovered.
H. B.	11 mos.	M.	Wet Nurse	2-16	2-17	2-19	{ Acute military tuberculosis. Enteritis.	Poor. Undersized. Anæmic.	Died.
F. S.	4 yr. 2 mo	F.	Regular	3-1	3-4	3-12	{ Enterocolitis.	Fair.	Recovered.
W. M.	4 yr. 4 mo	M.	Regular	3-30	4-2	?	{ None.	Fair. Rachitis.	Recovered.
P. C.	3 yrs.	M.	Regular	2-14	2-16	2-22	{ Bronchitis. Ac. croupous laryn. 2d rash (of rubeola), appeared, 3-13, disappeared, 3-18. Bron- chitis. Abscess of popliteal space.	Fair. Rachitical.	Recovered.
H. A.	4 yr. 3 mo	M.	Regular	3-3	3-5	3-11	{ 2d rash (rubeola), appeared 4-4. None.	Good.	Recovered.
H. W.	3½ yrs.	M.	Regular	?	2-1	2-8	{ 2d rash (rubeola), 3-2. 2d rash disappeared 3-5. None.	Good.	Recovered.
R. B.	4 yrs.	F.	Regular						

TABLE SHOWING THE COURSE OF THE DISEASE IN 107 CASES OF RUBEOLA—Continued.

NAME	AGE	SEX	DIET	Month and Day of Invasion	Appearance of Rash	Disappearance of Rash	COMPLICATIONS	GENERAL CONDITION	RESULT
J. B.	11 yrs.	M.	Regular	3-1	3-4	3-9	{ 2d rash (rubella), 3-23. 2d rash disappeared 4-3. Marked enteritis with rubella.	Good.	Recovered.
C. B.	4 y. 11 mo	M.	Regular	?	2-10	2-17	{ 2d rash (rubeola), appeared 3-16. 2d rash disappeared 3-22.	Good.	Recovered.
S. G.	2 yr. 4 mo	M.	Regular	?	3-13	3-20	{ Otitis media suppur. 2d rash (rubella), appeared 4-2. None.	Good.	Recovered.
A. D.	6 yrs.	F.	Regular	?	3-22	3-26	None.	Good.	Recovered.
S. C.	6 yrs.	F.	Regular	?	?	?	None.	Good.	Recovered.
M. W.	6 yr. 2 mo	M.	Regular	3-5	3-7	3-14	None.	Good.	Recovered.
T. T.	5 yrs.	M.	Regular	3-4	3-5	?	None.	Good.	Recovered.
W. H.	5 yr. 7 mo	M.	Regular	3-2	3-5	3-10	{ 2d rash (rubeola), appeared 3-19. 2d rash disappeared 4-2. None.	Good.	Recovered.
W. H.	6 mo.	M.	Bottle	2-?	?	?	Broncho-pneumonia.	Fair.	Sent to N.B.I. Died.
J. D.	8 yrs.	M.	Regular	3-24	3-27	?	None.	Good.	Recovered.
T. T.	7 yrs.	M.	Regular	?	?	?	None.	Good.	Recovered.
L. McC.	5 yrs.	F.	Regular	3-1	3-2	3-12	None.	Pott's disease.	Recovered.
J. F.	18 mo.	M.	Regular	2-8	2-17	3-8	{ Bron.-pneu. Pleurisy with effusion. Enteritis.	Poor. Thin. Not well nourished.	Recovered.
N. J.	1 yr.	M.	Breast	?	2-14	?	Enteritis.	Very poor. Negro. Marked rachitis.	Recovered.
J. E.	8 mos.	M.	Breast	?	2-25	3-7	Enteritis.	Fair.	Recovered.
R. D.	19 mos.	F.	Regular	3-10	3-13	3-23	Bron.-pneu. Enteritis.	Poor. Rachitis.	Recovered.
L. K.	18 mos.	F.	Regular	2-23	2-24	3-5	Bron.-pneu. General tuberculosis.	Fair.	Died.
M. R.	15 mos.	F.	Bottle	3-1	3-3	3-6	{ Bron.-pneu. Purulent synovitis of hip.	Fair.	Died.
A. B.	12 yrs.	M.	Regular	2-12	2-15	?	None.	Good. Epileptic.	Recovered.

TABLE SHOWING THE COURSE OF THE DISEASE IN 8 CASES OF PRIMARY RUBELLA.

NAME	AGE	SEX	DIET	Month and Day of Invasion	Appearance of Rash	Disappearance of Rash	COMPLICATIONS	GENERAL CONDITION	RESULT
F. L.	10 yrs.	F.	Regular	?	?	?	None.	Good.	Recovered.
T. G.	6 yrs.	M.	Regular	3-24	3-26	3-30	None.	Good.	Recovered.
W. B.	3 yr. 5 mo	M.	Regular	3-27	3-30	—	None.	Fair. Pott's disease.	Recovered.
L. H.	8 yr. 2 mo	F.	Regular	3-21	3-24	3-28	Acute tonsillitis.	Good.	Recovered.
G. M.	4 yr. 7 mo	M.	Regular	?	3-30	4-2	None.	Fair.	Recovered.
L. P.	4 yr. 3 mo	F.	Regular	?	3-18	?	None.	Good.	Recovered.
J. P.	4 yrs.	M.	Regular	3-4	3-6	?	None.	Good.	Recovered.
F. H.	6 yrs.	F.	Regular	3-26	3-28	3-31	Lobar pneumonia.	Good.	Recovered.

Lobar pneumonia, pertussis, and varicella,	1
“ “ very rhachitic,	1
“ “ membranous laryngitis. Very poor condition,	1
Gastro-enteritis and general rhachitic condition,	1
Chronic meningitis and marasmus,	1
General military tuberculosis,	1

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The pneumonia in three of the patients was probably a mixed infection. The symptomatology and physical signs were those of an acute lobar pneumonia. The onset was sudden, the area of inflammation was localized and distinct with the temperature range incident to a lobar pneumonia but the resolution was slow and unsatisfactory and all of the cases succumbed. One of these patients had a pleurisy with effusion. It is unfortunate that autopsies were not allowed.

POST-MORTEM examinations were made as follows:

AUTOPSY NO. 1. Richard Roche, aet. one year. Body fairly well nourished.

LUNGS.—Right pleural cavity contains one-half ounce of thick yellowish-green fibrinous pus, coating the lung, costal pleura and diaphragm. Left cavity normal. Right lung; lower lobe almost solid, broncho-pneumonia, sub-pleural abscess size of a small pea; middle lobe, partly aerated, posteriorly a cavity with softened, gangrenous walls, cavity without any pus and not opening externally. Upper lobe shows areas of pus infiltration, intense bronchitis, congestion and œdema. Left lung; lower lobe posteriorly shows areas of consolidation, congestion and œdema.

SPLEEN.—Congested.

LIVER.—Fatty.

KIDNEYS.—Markings pale, cortex thin, capsule free.

STOMACH.—Normal.

INTESTINES.—Not examined.

AUTOPSY NO. 2.—Pincus Brown, aet. eight months and fourteen days. Autopsy by Dr. Wollstein. Body, badly nourished, no skin lesions.

LUNGS.—No pleurisy; right lung intensely congested, at apex small area recent broncho-pneumonia, rest of lung well aerated, base congested and œdematous. Left lung; œdema and congestion of upper lobe, strip of broncho-pneumonia along posterior border lower lobe.

HEART.—Normal, foramen ovale closed; other organs normal.

AUTOPSY No. 3.—Harry Bliss, aet. nine months. (Wet nursed by mother of Mary Constable.) Autopsy by Dr. Wollstein. Body fairly well nourished.

BRAIN.—Pia cloudy and slightly thickened over cortex, most marked over base, tubercles along vessels, some at cortex, new tubercles in island of Reil and inter-peduncular space both sides.

LUNGS.—Recent fibrous pleurisy over right lower lobe posteriorly and between the lobes of the right lung; recent miliary tubercles on the surface of both lungs, most marked in lower lobe, large area of cheesy pneumonia in same lobe; left lower lobe atelectatic; bronchial lymph nodes large and cheesy, especially on right side.

SPLEEN. Enlarged, miliary tubercles on surface and in substance.

LIVER.—Tubercles throughout.

KIDNEYS.—One tubercle in cortex of right kidney, markings distinct; supra-renal capsules normal.

STOMACH.—*Post-mortem* softening of cardiac end and the surrounding area.

INTESTINES.—Colon, enlarged solitary follicles, congested near ileo-cæcal valve, a few young ulcers; ileum, Peyer's patches swollen and congested; jejunum four or five round tubercular ulcers.

MESENTERIC LYMPH nodes are swollen and contain tubercles.

PANCREAS, normal.

Acute general miliary tuberculosis.

AUTOPSY No. 4.—Mary C. O'Conner, aet. thirteen months. Body well nourished. No lesions of skin. *Post-mortem* ecchymoses.

LUNGS.—About eight ounces of sero-purulent fluid in left pleural cavity. Fibrinous and purulent exudate over pulmonary and costal pleura. Lower lobe of left lung solidified, broncho-pneumonia with purulent infiltration; upper lobe consolidated in areas.

RIGHT LUNG.—Pleural cavity contains two ounces of clear serum. There is no pneumonia but intense congestion. Emphysema anteriorly.

HEART.—Normal.

SPLEEN.—Large, firm and congested.

LIVER.—Congested.

STOMACH.—A little congested around œsophageal end, very thick mucus over walls.

INTESTINES.—Not examined except the colon which is normal except for hyperplasia of follicles.

KIDNEYS.—Large and pale, capsules free. Markings very indistinct, cortex thinner than normal, exudative nephritis.

POST-MORTEM DIAGNOSIS.—Broncho-pneumonia with purulent infiltration, emphysema, pleurisy, acute diffuse nephritis.

AUTOPSY No. 5.—Lillie Kenny, aet. eighteen months. Body fairly well nourished with *post-mortem* ecchymoses.

THORAX.—Pleuræ, no adhesions, no fluid.

LUNGS.—Left, upper lobe emphysematous and congested, small areas of atelectasis; lower lobe almost solid, broncho-pneumonia, pus in bronchi, softened in places, some small cheesy tubercles. Right upper lobe solid, broncho-pneumonia with purulent infiltration; middle lobe emphysematous anteriorly, few tubercles; lower lobe consolidated, large tubercles, cheesy on section, superficial atelectasis of diaphragmatic surface.

HEART.—Right border extends almost to mid-clavicular line, apex in the fifth space on the mid-axillary line. Pericardium normal, fluid a little increased. Structure of heart normal. foramen ovale closed.

LIVER.—Pale, no tubercles.

SPLEEN.—Normal, no tubercles.

STOMACH.—Normal but apparently enlarged, greatest length six inches, breadth four and one-half inches.

INTESTINES.—Normal.

KIDNEYS.—Normal, but congested, capsule free.

POST-MORTEM DIAGNOSIS.—Broncho-pneumonia with purulent infiltration, tuberculosis.

AUTOPSY No. 6.—Marguerite Rodriguez, aet. fifteen months. Autopsy by Dr. Wollstein.

LUNGS.—Left area of old pleural thickening at base, no recent pleurisy, thickening of bronchial walls, intense congestion and œdema, posteriorly a small area of consolidation, upper lobe well aerated. Right upper lobe well aerated: lower lobe, emphysematous anteriorly, some œdema and congestion.

HEART.—Normal, foramen ovale closed.

SPLEEN.—Large, soft, some perisplenitis.

LIVER.—Large, moderately fatty.

KIDNEYS.—Pale, markings indistinct, cortex not thickened.

STOMACH.—Normal.

INTESTINES.—Colon, solitary follicles enlarged, no ulcers in ileum, Peyer's patches slightly hyperplastic, no ulceration.

PANCREAS.—Normal.

LYMPH NODES.—Mesenteric normal. One bronchial lymph node on right side is enlarged and shows small yellow tubercles but no breaking down.

HIP JOINTS.—Left side normal. Right hip joint contains several drachms of yellow, rather thick pus. The abscess has not burrowed outside of joint. Acetabulum and head of femur covered with pus but not eroded.

BRAIN.—Pia mater cloudy at base, but otherwise normal.

AUTOPSY No. 7.—Mary Constable, aet. eight months. Body fat, well nourished.

LUNGS.—Right, few pleural adhesions, new process, about two drachms purulent white fluid on diaphragmatic surface. Left, two drachms of bloody serum in pleural sac. Right lung, upper lobe emphysematous anteriorly with translucent tubercles scattered over pleura and in its structure, a few cheesy small nodules show on section, tubercles all through lobe which is congested; middle lobe congested, tubercular and emphysematous, lower and anterior portion atelectatic; lower lobe consolidated posteriorly with broncho-pneumonia, posteriorly a small cheesy cavity the size of a hazel nut opens into a bronchus. Left lung, emphysematous and congested with scattered translucent tubercles; posteriorly the lower lobe shows broncho-pneumonia.

HEART.—Right auricle engorged and dilated, contains pale blood clot, foramen ovale a pin-point opening, fine tubercles on pericardium, valves normal, muscle pale.

LIVER.—Extremely fatty, no tubercles seen.

SPLEEN.—Enlarged, small tubercles on surface and scattered through it.

STOMACH.—Normal.

KIDNEYS.—Cloudy swelling, capsule free, cortex not thickened; right supra-renal capsule has a small cavity in centre with few small tubercles.

INTESTINES.—Ileum normal to two inches above ileo-cæcal valve where there is a pseudo-membrane extending through

to colon, follicles thickened and show commencing loss of substance. Colon, mucous membrane thickened and covered with a pseudo-membrane.

MESENTERIC LYMPH NODES are enlarged, pale on section but not cheesy. Bronchial and mediastinal lymph nodes enlarged, pale and cheesy, but not broken down.

BRAIN.—Pale, pia mater glistening, some fibrinous exudate beneath pia over brain cortex but no tubercles to be seen except a few along the blood-vessels on inner and upper surface of left hemisphere. Choridal plexus normal.

Thanks are due to Drs. E. Sturgis, E. Bulkeley, and F. O'Neil of the Staff for their care of the patients, and for the figures given in this report. Dr. E. G. Bryant of the Health Department, kindly furnished the statistics of the children transferred to the Health Department.

68 WEST FIFTY-FIRST STREET.

TERATOMA OF SACRUM AND COCCYX.

BY FRANCIS HUBER, M.D.,

Visiting Physician to Gouverneur Hospital; Chief of Clinic, Department
Diseases of Children, Vanderbilt Clinic; Instructor in Diseases
of Children, Medical Department, Columbia University,
New York.

In the transactions of this Society for 1892, I reported a case of sacro-coccygeal tumor in a child three weeks old; operation; recovery.

These cases appear to be as rare as they are interesting, and I am rather fortunate in being able to add another operated upon on the thirty-fourth day after birth, with recovery. The careful and thorough microscopic examination of the growth by the accomplished microscopist and pathologist, Dr. James Ewing, assistant in the Pathological Laboratory of the Medical Department of Columbia University, lends additional interest and importance to the case.

A proud nurse carrying an infant but a few hours old, attended by a consequential interpreter, with the abashed father in the background, formed an interesting tableau in the reception room of the Children's Department at the Vanderbilt Clinic. In answer to my questions as to the nature of the trouble, the only response I could get was—"Want him cut off!" The newly born infant, dressed in the regulation Italian style, was ordered to be stripped, and after some little delay, the reason for the reply, "Want him cut off!" was apparent—a congenital sacro-coccygeal tumor was disclosed.

The general characteristics of the growth are very well shown in the accompanying photographs. The child otherwise was well nourished and in good condition; no club-foot or interference with the development of the lower extremities. The growth involved the sacral and coccygeal regions, was centrally situated, the greater part of the mass, however, being to the left of the median line. Length posteriorly was four and one-half inches; anteriorly, two and three-quarter inches; slight con-

striction at base; diameter at this point, one, one-half and two inches; of tumor proper, two and three-quarter inches.

Digital examination failed to show any malformation of the anus or rectum, the rest of the intestinal tract evidently normal, as the first escape of meconium occurred during the examina-



Fig. 1. TERATOMA OF THE SACRUM AND COCCYX.

tion. Posterior to the anal orifice, the skin appeared to be normal for about an inch, then became continuous with the integument over the tumor, and adhering firmly to the underlying parts.

The greater portion of the tumor, particularly the lower two-thirds, was cystic in character, while towards the base the structures gradually became firmer, and were intimately and firmly adherent to the deeper bony structures. On the left side anteriorly and above (well shown in the photograph), was found a hairy mole, or rather swelling, about one inch base; on the other side, quite a large port-wine mark existed.

In the absence of Prof. Jacobi, the case was admitted to my service at Gouverneur Hospital. My sincere thanks are tendered to Dr. Jacobi for the case and opportunity to present the specimen with the microscopic report.

Operation November 18, 1897. Chloroform narcosis. Parts rendered aseptic. A triangular flap, base about one and one-half inches wide, with apex downwards, was dissected away from the anterior aspect of the growth; then a second incision, semi-lunar in shape, encircling the somewhat constricted base was made, after which the deeper structures were cautiously dissected away—due care being taken to hug the tumor closely. The greater portion of the intimate fibrous attachments to the sacrum were cut. It was found that the coccyx and small part of the lower portion of the sacrum were so intimately adherent that these structures were resected.

The dissection was necessarily slow, as the rectal walls were in close opposition to the growth. At the inner sacral aspect three small cysts were found, and along the anterior surface of the sacrum on the left side when the bone had been taken away, a thick, dense, prolongation, running upwards, was discovered. Its nature was not evident, and not being able to trace it up any great distance without wounding important structures, it was thought best to ligate and cut below the ligature. No communication with the spinal canal could be made out. But one vessel required ligature, when the coccyx was taken away, probably the coccygeal artery. At the bottom of the large wound the rectum could be seen for an inch. The proximity of the wound to the anus and the inability to obliterate the dead spaces entirely, made it advisable to pack lightly with iodoform gauze. The greater part of the skin wound was brought together transversely with catgut sutures, the apex of the anterior flap being attached to the skin over the sacrum and a small portion on left side left open to allow removal of the

gauze. Dressing then applied and patient sent to ward in excellent condition.

The subsequent progress was uneventful; the child was quieted by small doses of paregoric, and kept lying on its



Fig. 2. TERATOMA OF THE SACRUM AND COCCYX.

stomach to prevent soiling of dressings. The greater part of the wound healed primarily; the cavity lightly packed with gauze was allowed to granulate. Patient discharged cured December 19, 1897.

The insignificant character of the scar is well shown in the photograph taken when the child was seven months old.

In speaking to a very prominent surgeon in regard to the case, he remarked that it was interesting to him, inasmuch as it showed how well infants tolerated severe surgical interference. The absence of shock is attributed in great measure to the skilful manner in which the chloroform narcosis was conducted by Dr. Jos. Huber.

My thanks are tendered to Dr. Pafford, House Surgeon, Gouverneur Hospital, and to the members of the staff, as well as the nurses, for the attention given the case.

MICROSCOPICAL EXAMINATION: By Dr. James Ewing.—The main mass of the tumor is composed of a granular, semi-fluid material, which on section proves to consist of imperfectly developed *nervous tissue* containing bipolar and multipolar nerve cells, neuroglia cells, neuroglia, and non-medullated nerve fibres. These elements are massed together very irregularly, but often reproduce the convoluted appearance of the cerebral gray matter. Stained with methylene blue, the chromatic substance and nuclei of these cells are seen to be very imperfectly developed or entirely absent. The central areas of the nervous tissue are largely necrotic. Along the periphery it pushes its way for considerable distance among the other tissues of the tumor, but is everywhere separated from these tissues by a layer of endothelial cells. Throughout this mass are many irregular spaces of considerable size lined by *cuboidal epithelium* identical in appearance with the lining cells of the central canal. Some of these spaces are lined, however, by long *cylindrical epithelium* which sends numerous branching processes into the adjacent nervous tissue and recalls distinctly the appearance of *embryonal neuroblasts*. Along the edges of other spaces are many true ganglionic cells, some of which resemble the Purkinje cells of the cerebellum.

In sections through the pedicle, the spaces lined by cuboidal epithelium are abundant, and some appear to be continuous with the central canal of the spinal cord.

Surrounding the central mass of the tumor are a number of embryonal structures supported by ordinary connective tissue. Near the base of the tumor are several masses and plates of *embryonal hyaline cartilage*. These are surrounded by connective tissue in which there are some lobules of fat cells, and a few

glandular alveoli of the type of mucous glands. In the same vicinity are several large spaces lined by cylindrical *ciliated epithelium*, and the association of these structures suggests an attempt to reproduce in the tumor the *respiratory passages* of the embryo.

In the neighboring region of the tumor were seen some elongated spaces lined by high columnar epithelium identical in ap-



Fig. 3. DR. HEWLETT'S CASE OF CONGENITAL SACRO-COCYGEAL TUMOR.

pearance with that found in the ducts of the mucous glands of the stomach, and representing the element of the *gastro-intestinal tract* which is usually to be found in teratomata.

A few bundles of a striated voluntary muscle fibres were found at several points in fibrous tissue.

A prominent excrescence projecting from the base of the tumor was found to be composed exclusively of fat tissue.

The entire tumor was surrounded by the distended integu-

ment of the buttock, and presented no abnormalities of interest.

J. Bland Sutton, in his usual careful and scientific manner, has made a study of the tumors found in the sacro-coccygeal region. His classification has been referred to in the report of my first case in the transactions for 1892.

The careful and thorough microscopic examination made by Dr. Ewing, leaves no doubt as to nature of the tumor, and places the specimen presented to-day in the category of teratomata. A teratoma as defined by Sutton is an irregular conglomerate mass, containing the tissues and fragments of viscera of a suppressed foetus, attached to an otherwise normal individual.

To appreciate the nature of these anomalies, Sutton found it necessary to consider the subject of conjoined twins, supernumerary limbs and acardiac foetuses. I shall take the liberty to present his conclusions taken from his work "Tumors, Innocent and Malignant."

"Thus a study of the circumstances surrounding the development of twins and duplex monsters brings us to the conclusion that teratomata may arise either from partial dichotomy of the trunk axis of the embryo, or from complete dichotomy. In the latter case, while one twin has gone on to full development, the growth of the other has been arrested, and in some cases the suppression has been so great, that the companion foetus is represented by a deformed or shapeless mass consisting of integument covering ill-formed pieces of the skeleton and portions of viscera.

"In a few cases of parasitic foetuses we are able to offer a probable opinion as to whether the reduplicated parts are due to partial dichotomy of the trunk or are the result of complete cleavage, in which one of the foetuses becomes an acardiac. In very many, indeed in the majority of teratomata, it is absolutely impossible to decide in favor of one method or the other.

"TREATMENT.—Parasitic acardiacs are in almost all cases so extremely valuable as sources of gain in fairs, shows, and large cities, that the parents, or the unscrupulous individuals who get possession of these children, will not permit operative interference. When the parasitic acardiac is of the amorphous va-

riety and attached to the dorsal surface of the sacrum, attempts may be made to remove them. The children rarely survive the interference."

In this connection, the following case may be of interest, as representing another variety of the tumors met with in this situation:

At the invitation of Dr. W. W. Hewlett, of Babylon, N. Y., to whom I wish to tender my thanks, I was permitted to examine a little patient seven months old, with a sacro-coccygeal tumor. The growth presented a uniform shape and was evidently a unilocular cyst, situated between the rectum and the anterior sacral surface and coccyx. Its general appearance is distinctly shown in the illustration (Fig. 3). How far it extended upwards in the pelvis could not be determined, as the anal sphincter was tense, firm and would not yield or dilate, upon attempted introduction of the finger.

In the absence of an exploratory puncture with a careful examination of the fluid contents, a differential diagnosis between post-rectal dermoid and anterior sacral spinal bifida was not made.

The skin was normal in every respect, no pigmentation, excessive growth of hair, or other abnormality visible. Fluctuation distinct; no translucency. The fatty tissue in excess formed a diffuse lipoma over the cystic growth.

No abnormality present along the spine further up; no evidence of chronic hydrocephalus. Aside from the cranio-tabes and other evidences of rickets, child well.

The presence of the diffuse lipoma would lead me to infer that the cyst below is probably connected with the spinal canal and that we have to deal in this case, with a spina bifida. To make a positive diagnosis, a careful chemical examination of the fluid should be made.

The history of the case furnished by Dr. Hewlett is briefly as follows: Mary M., born of healthy parents, weighed at birth ten pounds, has a large tumor of a soft semi-elastic feel, attached to the inferior and posterior extremity of the trunk. The growth does not interfere with nutrition nor any healthy function. She is now aged nine months, weighs seventeen pounds and is in good general health. The relative size of the tumor does not

vary perceptibly from its appearance at birth. She measures $14\frac{1}{2}$ inches around the abdomen on line with umbilicus, and $16\frac{1}{2}$ inches around hips and most prominent part of tumor. Base of tumor 14 inches in circumference; from tip of coccyx to anus $6\frac{3}{4}$ inches.

The accompanying photograph was taken when the child was four weeks old.

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DISCUSSION.

DR. JACOBI.—I would like to ask in which direction did that duct or string go up?

DR. HUBER.—Along the anterior sacral aspect. After the coccyx and sacrum had been gotten out of the way, there was a sort of prolongation that ran up. In the literature I find that very common.

DR. JACOBI.—Was it a string or a duct?

DR. HUBER.—It was difficult to say which it was. It was a sort of fibrous band that it was thought best to ligate and cut.

DR. JACOBI.—The doctor gave Sutton's division of these tumors. What did I understand him to say in regard to Luschka's gland?

DR. HUBER.—He considers it only the remains of a fetal condition in the post-anal gut.

DR. JACOBI.—That is a mistake on Sutton's part. Luschka's so-called gland, it is true, is not a gland at all; that has been proven long ago, but Luschka's gland now, I think, is established as being principally composed of blood-vessels, the last peripheral ramification of the middle sacral artery. They have been injected, which is ample proof of its vascular nature. Between those little branches, five or six generally, there is connective tissue and also nerve fibres, mostly sympathetic. That is what Luschka's gland is. Therefore it is possible that tumors of a fibromatous kind and of an angiomatous kind, now and then even sarcoma, may take origin in this so-called "gland." Other tumors, that were formerly attributed to this source, particularly by Braune in his large Atlas on Sacral Tumors, must not be attributed to this so-called Luschka's gland. Now the

question is where this came from. This tumor is really a teratoma, inasmuch as it consists of ever so many different tissues which are found in one mass. This tumor contains cartilage, fluids, intestinal mucous glands and respiratory organs, so that it contains in fact, with the skin, all such tissues as come from the different germinative layers. The ectoderm and the mesoderm and the entoderm are all represented in that tumor. Now there is one point where all those meet each other in early embryonal life, viz. : the lower end of the early nervous system, which is probably just the point that these tumors come from, and it appears that an abnormal connection between the three might be considered the original cause of the tumor. These cases are usually attributed to the implantation of another fœtus. It looks to me as if this might be a case of development arrested at an early period when the three germinative layers are closely packed together.

CASES OF TUBERCULAR MENINGITIS WITH PECULIAR
RESPIRATORY PHENOMENA.

BY HENRY DWIGHT CHAPIN, M.D.,

New York.

The irregularity and varied character of the symptoms of tubercular meningitis are well known to all observers of this disease. They may be partially explained by the fact that it is rare for the meninges alone to be attacked by the tubercular process, which is usually of a diffused miliary character. In very young subjects the bronchial glands and lungs are nearly always involved. The symptoms produced by this involvement of various vital organs are surprisingly few when the meningitis assumes a pronounced form. The respiration is nearly always affected, assuming an irregular, sighing character, and when the disease is well developed the Cheyne-Stokes variety is apt to be noted. The object of the present paper is to call attention to a peculiar and intense form of dyspnœa that may rarely be seen in connection with severe cases of tubercular meningitis. As far as the writer can find, very little attention has been paid to this peculiar and distressing symptom.

CASE I.—Alice K., ten months old, admitted to the Babies' Wards, December 26, 1897; died January 3, 1898. *Family History*: Both parents living and in good health. Baby was born healthy and has had no illness except a slight diarrhœa during the summer. *Present Illness*: Three weeks ago the child began vomiting and became very restless. Vomiting occurred once or twice a day after feeding. A slight cough developed. Bowels were constipated. Four days ago the baby was suddenly seized with general convulsions. Since then has had repeated

attacks, as many as fifteen a day. *State on Admission:* Well developed baby; body rigid; eyes staring and pupils dilated. Examination of the lungs negative.

PROGRESS OF THE CASE.

December 26th.—Bowels irrigated; return flow brought away a quantity of green and yellow fæcal matter with mucus. Temperature, 100°; respiration, 48; pulse, 125. Several slight convulsions on the right side during the night; one general convulsion lasting three-quarters of an hour at 10 P.M.

December 27th.—Constant twitching of right leg all day. One general convulsion in the afternoon lasting about twenty minutes. Lumbar puncture was performed and half a drachm of clear fluid removed. No tubercle bacilli found after careful examination by bacteriologist. Child fed by gavage. Slept well during the night but constant twitching of the right leg is present.

December 28th.—Had two general convulsions during the day. Pupils of both eyes very much contracted for about an hour in the morning. Arms very rigid for the greater part of the day, with occasional twitching of right leg. Examination of the eyes showed beginning papillitis of the right eye; left eye normal; pupils sluggish. Remained rigid all night and had one general convulsion.

December 29th.—Slept all the morning. At times rigid, respiration becoming very irregular. A general convulsion at noon. A number of slight convulsions during the night, two lasting about half an hour. Respiration and pulse both irregular.

December 30th.—Constant twitching of the right leg. Rolling of the eyes and the right eye turned in all day. Pulse very rapid and irregular. During the night the left arm and leg were in a spastic condition. Right eye turned inward. Profuse perspiration about the head and did not sleep as well as usual; kept sighing and moaning.

December 31st.—Remained rigid the greater part of the morning. Respiration irregular and shallow. Pulse stronger but very rapid. During the night remained rigid, with very few convulsions.

January 1, 1898.—Slept greater part of the morning; twitching of eyelids when awake. During the evening the

respiration became irregular and slow. At midnight pulse was stronger and respiration more regular.

January 2d.—At 2 A.M. respiration became very labored with great dyspnoea on inspiration. Air entered the lung very imperfectly. During inspiration the chest-wall sank in to a marked degree and the child seemed about to die from dyspnoea. This continued in varying degree until death in the afternoon. Coarse, moist râles were heard all over the chest. For twenty-four hours before death the child showed all the symptoms of severe membranous croup. During the illness the temperature varied from 100° to 103° F.; but just before death it arose to 106.6° F. The respiration varied from 20 to 54, and the pulse from 120 to 160. Examination of the urine showed hyaline casts but no albumen nor sugar.

AUTOPSY—BRAIN.—Skull-cap thin; excess of fluid about the brain. There is marked fibro-purulent exudation on the under side of the brain, especially at the pons, medulla, and optic commissure. There are small scattered miliary tubercles throughout this area; also on the inferior surface of the frontal lobe about the olfactory bulb, and on the inferior and superior surface of the cerebellum. Both surfaces of the latter are studded with tubercles, most marked on the upper surface. There are also tubercles along the fissure of Sylvius. The convex surface of the brain presents a smooth, glistening appearance with a few small tubercles on both sides, and with markedly distended blood-vessels. The lateral ventricles contain an excess of rather turbid fluid. The walls are very soft and easily broken down. A few tubercles can be seen on the choroid plexus but none are noted in the lining of the ventricles.

CHEST.—The bronchial glands enlarged and caseous. Lower portion of the lobe of the left lung shows on section a cheesy tubercular area; rest of the lung fairly well aerated. Bronchi are congested and coated with mucus. There are adhesions between the upper and middle lobe of the right lung and congestion and œdema of the lung, but no tubercular areas.

Other organs normal.

Diagnosis, acute tubercular meningitis, chronic tuberculosis of bronchial glands and of the left lung.

CASE II.—Hulda E., four years of age, admitted to the Babies' Wards, November 27, 1897; died December 8, 1897. *Family History:* Both parents living and healthy; five other

children who are healthy. The child was breast-fed; had measles three years ago and whooping-cough last spring. *Present Illness*: Two weeks ago while playing under a table, she arose suddenly and struck her head severely against the under side of the table. No abrasion of the scalp was noted. Four days later, she began to complain of severe headache. She vomited all her food and some green matter. The vomiting stopped after four days. When left alone the child closes her eyes and wishes to sleep, at times awakening to cry out with pain. Has been feverish at times. *State on Admission*: General appearance good. Heart and lungs negative. Weight, 37 pounds; respiration, 28; pulse, 128; temperature, 98° F.

November 27th.—No stool during the day; dose of calomel administered. Was noisy at night and did not sleep well. Took considerable nourishment.

November 29th.—Complained of pain in the head.

December 1st.—No stool in forty-eight hours; enema given. Examination of the eyes showed pupil of the left eye more widely dilated than that of the right; fundi normal; both eyes turned toward the left most of the time.

December 2d.—Eyes slightly blood-shot; pupil of left eye very much dilated. No movement of the right arm noticed during the day.

December 3d.—Could with difficulty be roused to take nourishment. Soon went into a comatose condition and remained so all day. When roused for nourishment seemed unable to open the left eye. No movement of the right leg noticed. She remained in a comatose state all night and refused nourishment.

December 4th.—Some congestion of the fundus of each eye, but no actual inflammation. Pupils contracted, but the left still a little more than the right. The conjugate deviation noticed three days ago is now absent. Remained in comatose state all day. Breathing was noisy during the day but more quiet at night.

December 6th.—Appeared somewhat better. Pulse stronger and respiration more even. Right pupil very much larger than the left. Took nourishment better than usual.

December 7th.—Lumbar puncture performed and clear fluid withdrawn. Bacteriological examination showed no tubercle bacilli or other germs. During the morning she was seized with great difficulty in inspiration, all the accessory

muscles of respiration being brought into play. There was a sucking in of the soft parts on inspiration and dropping of the larynx. Color fairly good. Breathing seems to be entirely thoracic. The air seemed to enter better when she was in an upright position. The dyspnœa increased to such an extent that a tube was inserted which seemed to somewhat improve the breathing. Patient was fed by gavage. In two hours the tube was withdrawn. The child presented all the evidences of diphtheritic croup in an extreme stage. A throat culture was taken from the mouth and found to be free from Klebs-Loeffler bacilli. The dyspnœa seemed to come in an irregular manner, at times being much worse than at others.

December 8th.—Air enters the lungs very imperfectly, but appears to enter the left side much better than the right, with the disparity more marked at the apices. Death finally ensued from exhaustion rather than from dyspnœa.

The temperature varied from normal to 104° F.; on the day before her death it arose to 106.8° F. Respiration varied from 28 to 70, and the pulse from 95 to 170.

AUTOPSY.—No sign of injury apparent.

SPINAL CORD.—On opening the vertebral canal 25 c.c. of cloudy fluid escaped. There is a tubercular exudate on the surface of the dura mater and adhesions between the latter and periosteum of the vertebræ, especially marked at the upper portion and down as far as the fifth dorsal vertebra. The pia mater is slightly congested. On section of the cord no gross lesion is apparent. Substance is pale and very soft.

BRAIN.—Surface of the dura congested but smooth and glistening. Pia mater is intensely congested. There is a serous exudate in its meshes, in places compressing the brain, and flattening the convolutions. This is most marked over the anterior portion of the third frontal convolution and about the fissure of Rolando on the left side. The pia is thickened, especially at the base of the brain and over the superior surface of the cerebellum and in the fissure of Sylvius. In these regions were found small pin-head, translucent nodules, apparently miliary tubercles. The lateral ventricles each contained about 5 c.c. of clear, straw-colored fluid. The ependyma is smooth and glistening. The choroid plexuses are markedly injected and studded with small miliary tubercles. On section

the brain substance shows congestion. The basal ganglia, pons and medulla, show no gross lesions.

CHEST.—The left pleural cavity free from adhesions and smooth and glistening. On the right side in the axillary line between the first and second ribs are a number of firm, old plastic adhesions, otherwise the pleural cavity is free. The lungs are firmer than normal and everywhere through them can be felt small nodules. Between the pleura of both lungs are marked tubercles. On section miliary tubercles are found scattered throughout both lungs. The lower lobes are congested and œdematous. The bronchi are congested and contain considerable thick mucus of a light yellowish color. There are old adhesions between the lobes of the right lung, and in the lower portion of the upper lobe is a rather firm area about 3 c.c. in diameter, which on section shows a cavity nearly 1 c.c. in diameter. The inner surface of the cavity is covered with thin, purulent material. The wall is quite firm and surrounded by an area of consolidation. The bronchial glands are greatly enlarged and contain cheesy material in their centres.

ABDOMEN.—Intestines moderately distended. Omentum adherent to abdominal wall and to coils of the small intestine. The omentum is thickened and studded with miliary tubercles. The peritoneal surface of the intestines is covered in places with fibrinous exudate. There is no fluid in the pelvis. The dome of the diaphragm is unusually high, on the right side extending to the second rib and on the left side to a corresponding point to the third rib. Its under surface is studded with miliary tubercles. Mesenteric glands enlarged. Spleen is found studded with miliary tubercles. Liver is of usual size and its surface is dotted with tubercles. On section it is found dark in color but no tubercles are apparent. Kidneys are slightly enlarged; capsule smooth and non-adherent; cortical portion swollen, pale in color with indistinct markings; no tubercles found.

Other organs healthy.

CASE III.—As no autopsy was allowed in this case, a very brief account of it will be given.

Lillian W., eighteen months old, was sent to the Babies' Wards with the diagnosis of cerebro-spinal meningitis. She had had convulsions and presented all the symptoms of a general meningitis. She was taken with a croupy cough. Fearing an attack of diphtheria, fifteen hundred antitoxin units were

given and a culture taken. The throat showed no evidence of diphtheria, however. The report on the culture was negative. The croupy symptoms and dyspnœa increased and there was great difficulty on inspiration with sucking in of the soft parts. The dyspnœa did not become so extreme as to warrant intubation. The child did not die with symptoms of stenosis of the larynx but apparently from the usual course of the disease.

The temperature was most irregular during the illness, varying from 98° to 106° F., on six or eight different occasions going above 105° F. Respiration was also irregular from the beginning. Examination of the eyes showed that the discs were much too pale, as if there was an incipient atrophy. The blood-vessels were not much changed but the finer ones were apparently gone. There were convulsions during the course of the illness.

In comparing this case with the others, it has seemed to the writer that the meningitis was probably of a tubercular character and the dyspnœa of the same nature. It is difficult to explain exactly why these cases presented the evidences of extreme stenosis or obstruction to the entrance of air. In the first two cases the autopsy showed absolutely no membrane nor any apparent physical cause for this dyspnœa. Whether it was due to pressure upon the laryngeal nerves by the enlarged bronchial glands, or to some cause acting upon the respiratory centres in the medulla, it is difficult to say.

I am indebted to Dr. Davis for examining the eyes, and to Dr. LeWald for pathological work in these cases.

In the *Scottish Medical and Surgical Journal* for November, 1897, Dr. James Carmichael reports a case of basilar meningitis, not tubercular, in an infant aged five months, who died on account of attacks of difficulty in breathing, coming on especially at night. In reporting the case he states as follows:

“Chest is well developed. There is evident inspiratory difficulty. Expansion is free in its upper part and there is inspiratory recession in the epigastrium and in-drawing of the diaphragmatic insertion and lateral bases, producing a kind of hour-glass shape of the chest during respiration. Auscultation shows an old and harsh respiratory murmur without accompaniment except that of the loud snoring sound which is heard all over the chest.”

He further states: "The attacks of dyspnœa occurred at very irregular intervals. At times, especially when the baby is taking his bottle, the respiration is quite free and easy. When the attack comes on it is noticed that he makes a series of inspiratory efforts of increased force during which no air enters the chest and there is great lateral retraction of the thoracic walls and epigastrium. During these efforts to breathe he wriggles about and at last draws in a long gasp of air with a loud snoring sound and respiration is soon re-established, after which he generally has a fit of crying. During the attack the mouth is partially open and the tongue quite retracted, apparently shutting down the epiglottis. Forcible pulling forward of the tongue or depression of its base relieves his breathing to some extent although not completely. In one of these attacks the respiration completely ceased and the child became pulseless and appeared to be dead. Artificial respiration aided by a tube passed into the larynx restored the breathing." The patient gradually grew weaker and died. Autopsy showed a basilar meningitis. The larynx was found normal except a little catarrh behind the epiglottis.

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